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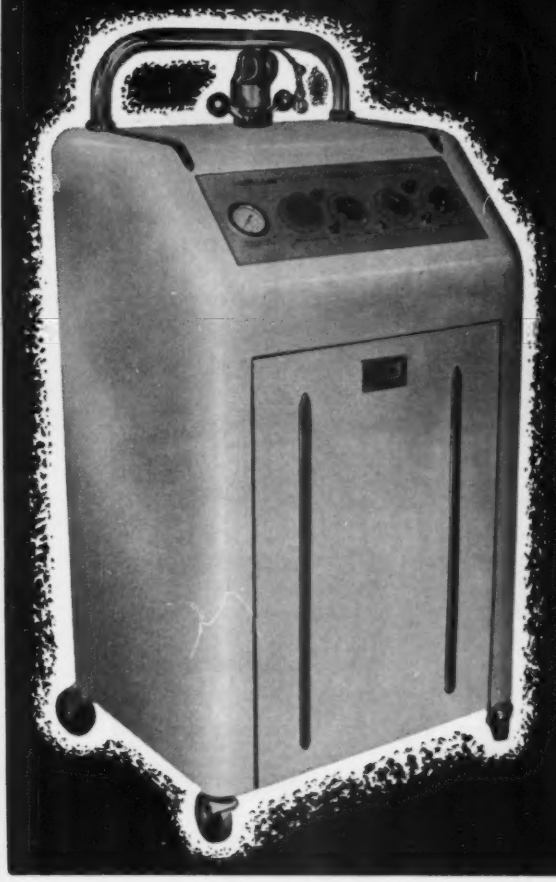

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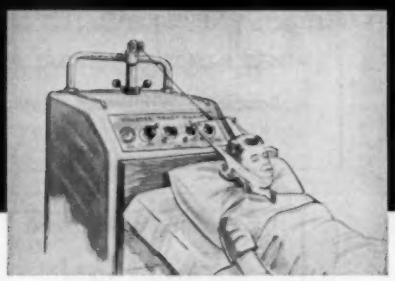

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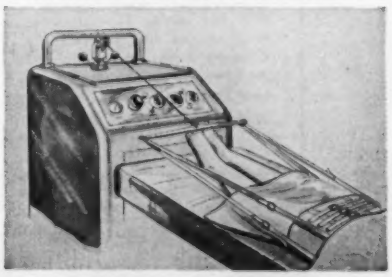

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LET'S PAUSE TO GIVE THANKS

Yesterday we were having a meeting to discuss the company's annual Thanksgiving gift to our employees. One of our Foremen mentioned ". . . it's a good thing the company thinks of a *gift* at Thanksgiving. Between Russia and taxes we don't have much else to be grateful for these days."

My inclination was to agree with him. "Why can't we have life like we used to in the 'good old days' ", I thought, "when everything was better, the money you made was your own, no H-bombs, life was worth living. . .". Later in the evening I got to thinking about it again. . . trying to determine just exactly *when* were these days I yearned for. I couldn't help but realize that history is made up of people yearning for the "good old days" . . . and that these days almost invariably were just a generation before.

To me, the "good old days" were back in the '20's. And yet I remember my father, in these same '20's, saying that times were tough and that he wished things were the same as they had been in the '90's. My grandfather, when I was young, used to tell me tales of the 1860's and 1870's . . . before the tough times of the '90's and after. And, someday, my daughter will think of *this* year as one of her "good old days" . . . as she tells her grandchildren about the "good old '50's and '60's."

I suppose it boils down to one thing; each generation, as it is faced with its own problems, secretly yearns for the time when life was less complicated . . . before it had to grow up and meet the challenge which has faced every human being since the birth of time. The circumstances may vary, but the question is always the same: "How can I live *my* life at peace with myself?"

"But *our* case is different" we say. True. Because each generation has to face different sets of problems. But, is paying my income tax worse than starving in the New England of 1620? Is the threat of Russia more immediate to me than an Indian war-cry to a desolate wagon train crossing Wyoming? Were the half-*million* Americans killed in the Civil War any more alive because of death due to "obsolete weapons"?

Yes, our case is different . . . but perhaps its problems are no more real or no more frightening than those which have faced every generation of Americans. And, perhaps, the answer is as it has *always* been . . . to face the problems of *now* with courage, so that the problems of tomorrow can be met on their own terms.

Yes, I think I *will* be grateful this Thanksgiving. Grateful to have been born in the greatest free nation in the history of mankind. Grateful that however dismal the future may appear, the solution lies within our determination to *face* our problems with all our experience, strength and hope. Grateful to be able to pray, as Reinhold Niebuhr wrote in 1935: *"God grant me the serenity to accept the things I cannot change, the courage to change the things I can, and the wisdom to know the difference."*

Happy Thanksgiving!

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The Importance of Rehabilitation in the Treatment of Chronic Pulmonary Emphysema

Albert Haas, M.D.
and
Aleksander Luczak, M.D.
New York

● The national incidence of chronic obstructive pulmonary emphysema is in excess of one million. Sufferers from this crippling disease are burdening the economy and seriously affecting the welfare of families. Usually they have been given only symptomatic relief with antibiotics and bronchodilators, which do not affect the progress of pulmonary changes.

It is not sufficiently recognized that rehabilitative measures are as important in the treatment of chronic obstructive pulmonary emphysema as in treatment of neuromuscular-skeletal disorders. Proper breathing exercises and technics affecting postural drainage have not been customarily used. There has been a general skepticism about these procedures, possibly because their effectiveness has not been scientifically demonstrated. Spirometric evaluation has been attempted, but was found not sufficiently sensitive for detection of significant changes following breathing exercises. The value of these measures to date has been determined solely by the subjective reactions of patients or the judgment of the clinician.

Obviously, if patients handicapped by pulmonary insufficiency can improve their breathing pattern, they are bound to make better use of their diminished cardiopulmonary reserve. Treatment which achieves this result is basic. Our own clinical observation led to the belief that intensive rehabilitative measures are of great value and justify laboratory study. Further, we found that improvement in pulmonary function after respiratory exercises can be readily determined by measurements of energy cost plus oxygen debt and evaluation of recovery.

Accordingly, a five-year study sponsored by the Office of Vocational Rehabilitation was undertaken at the Bellevue and Goldwater New York University Rehabilitation Services, in order to determine whether therapeutic breathing exercises can improve poor pulmonary function; what change in energy cost of activities of daily living occurs after therapy, with comparisons of oxygen debt and recovery in performance of these activities before and after treatment, and whether patients can be rehabilitated to a great degree of self-sufficiency, and realize vocational goals.

One of the most difficult problems in clinical medicine is the management of patients with chronic obstructive pulmonary emphysema. Recent surveys[1] have shown that more than one million persons are suffering from this crippling disease in the United States, burdening the economy of the nation and the families involved.

The cause of emphysema is only partly understood, and as a result opinions as to treatment vary, as Mitchell[2] pointed out.

The clinical course of chronic emphysema is characterized by periods of exacerbation and of quiescence and by relatively slow progression. Whittenberg and Ferris[3] stated that the end result is respiratory insufficiency, cor pulmonale and ultimately, perhaps, right heart failure. Patients with advanced pulmonary emphysema usually are severely handicapped. Rehabilitation of these patients, therefore, according to Rusk and associates,[4] is as important as rehabilitation of patients handicapped by neurologic or orthopedic disabilities.

Physical examination of the typical patient shows kyphosis of the upper part of the trunk, with fixed elevation of the shoulders, which gives almost the impression of orthopedic deformity. Evaluation of pulmonary function shows great intensification of respiratory effort, diminution of expiratory flow, decrease in vital capacity and timed vital capacity and increase in residual volume. Alveolar ventilation is impaired and uneven, and leads to arterial hypoxemia and increased carbon dioxide tension. The percentage of arterial oxygen saturation during rest is low as a rule, and rapid desaturation occurs with the slightest effort.

It is important that the intra-alveolar gas exchange be accomplished by the respiratory and circulatory systems with a minimum expenditure of energy. In patients with emphysema, adequate gas exchange can be achieved

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Read at the 3rd International Congress of Physical Medicine, Session on Balneology, Washington, D. C., August 24, 1960.

only by a marked increase in work done by the respiratory and accessory muscles. Whether or not maximal effort can produce adequate ventilation is the critical factor for survival.

Mechanically, breathing is accomplished 65 per cent by the diaphragm and 35 per cent by the accessory respiratory and intercostal muscles, according to Whittenberger and Ferris.[3] In the emphysematous patient, the expanded lung causes the diaphragm to be low and flattened and the costal wall to be "frozen" in a position of inspiration. Patients habitually assume a kyphotic posture, because this eases breathing during rest. However, McIlroy and Christie[5] observed that when oxygen demand is increased, this "hunchback" position greatly increases the work load in respiration. When the abdominal muscles are not used in normal respiration, they become flabby and atrophied, and this lessens the already low over-all efficiency of respiration. Weak abdominal muscles lack the strength to push the viscera upward, which is essential to effective diaphragmatic breathing in the emphysematous patients. Since emphysematous lung tissue loses its elasticity, the normally dome-shaped diaphragm is flattened in patients with chronic emphysema by the pathologically distended lung; therefore, its normal excursion in contraction (inspiration) is impaired to some extent. Inevitably the accessory respiratory and intercostal muscles are called on to assist inspiration, and breathing becomes increasingly laborious. The extra burden of breathing combined with the demands of routine activities of daily living results in prolonged oxygen debt, with a trail of consequences that cause damage to vital cells and embarrassment of metabolic functions. Therefore, patients with pulmonary emphysema have low tolerance to activity, and oxygen debt follows relatively moderate or even slight exercise, according to Margaria and associates.[6] This oxygen debt can be repaid only after prolonged cessation of activities.

Treatment

Treatment usually is centered on obtaining symptomatic relief with antibiotics, bronchodilators and oxygen with intermittent positive pressure breathing. These remedies bring some relief to the patient, but cannot, of course, influence the pathophysiologic pulmonary changes, which are irreversible. Postural drainage and therapeutic breathing exercises also are effective.

Physical rehabilitation of patients with chronic pulmonary emphysema may not be spectacular, yet it is as important as rehabilitation of patients with any other severe disability. Impressive results can be obtained with the respiratorily crippled. The objectives of respiratory exercises are to reeducate the patient to breathe properly by use of the diaphragmatic and the abdominal muscles, to improve body posture and to avoid any faulty tendencies. These exercises, diligently carried out, will increase the strength of the abdominal muscles and the excursion of the diaphragm, and tend to decrease the excessive burden of contraction of the upper intercostal and accessory respiratory muscles.

Evaluation of Breathing Exercise

To evaluate the importance of therapeutic breathing exercises, a research project has been conducted by the Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center, New York. A 15 bed unit was provided for this study at Goldwater Memorial Hospital. Over three years, 69 men and 12 women ranging in age from 28 to 60 have been treated. All patients had established obstructive pulmonary emphysema with cor pulmonale and in some cases right heart failure, described by McLean.[7] These patients had been hospitalized many times for this affliction, had been unemployed for long periods and required welfare support. On admission, they showed the characteristic symptoms: chronic coughing, difficulty in expectorating, cyanosis, shallow and rapid breathing, easy fatigability and dyspnea on slight effort in the routine activities of self-care. Pulmonary function tests showed significantly decreased vital capacity, increased functional residual capacity, greatly diminished maximum breathing capacity and an increase in the ratio of residual volume to total lung capacity, described by Comroe.[8] The spirometric tracing showed trapping of air. Studies of gas

in the arterial blood showed high carbon dioxide tension (from 50 to 70 milliequivalents per liter) and low oxygen saturation (from 86 to 60 per cent).

During the period of hospitalization for these patients (generally from eight to twelve weeks) determinations of pulmonary function and blood gas were made, and studies of oxygen consumption during activities requiring low, medium and heavy expenditure of energy also were carried out, with determination of oxygen debt and rate of recovery. Four activities involving varying "work loads" (Gordon[9]) were utilized: loop weaving in a sitting position, "floor loom" weaving in a sitting position, walking on a level and stair climbing. The open circuit Douglas bag method was used to collect the expired gases, which were analyzed by the Scholander method (standard temperature —pressure—dry). Oxygen consumption was first measured with the patient at rest in a sitting position. This was followed by three minutes of work, to give the patient an opportunity to reach physiologic equilibrium (steady state). Then five minutes of work was done, and the expired oxygen and carbon dioxide were evaluated. When the task was completed, patients were allowed to return to a resting position, and the expired gases were collected for thirty minutes to determine the oxygen debt and the time of recovery.

Psychosocial and vocational evaluations were carried out during the hospitalization in order to determine the probability of the patients' eventual reintegration into society and their independence in self-care. After discharge, the patients were followed at regular intervals in the out-patient department, and periodic studies of pulmonary function were made.

Fifty out-patients with pulmonary emphysema served as a control group. The majority of these patients were unemployed and were supported by the Department of Welfare. These patients received only symptomatic remedies (bronchodilators, oxygen with intermittent positive pressure breathing valve and antibiotics[10]), but not rehabilitation measures (postural drainage and breathing exercises). Periodic studies of pulmonary function were made of these patients also.

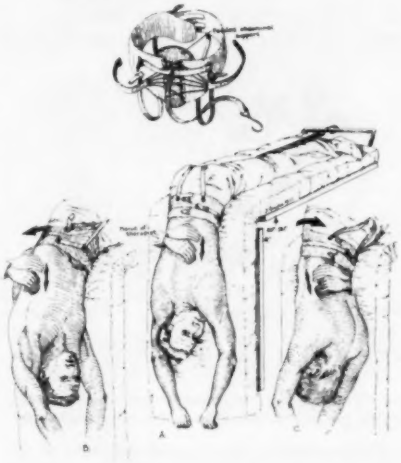

Fig. 1 — *A, B, C,* proper positioning for maximal effectiveness in postural drainage.

For the group of 81 hospitalized patients, clinical and laboratory studies were carried out, and treatment was begun with bronchodilators, antibiotics, a detergent and intermittent positive pressure breathing with oxygen. To help clear the bronchial tree of secretions that could not be raised by coughing, the patients were subjected to postural drainage, which preceded the therapeutic exercises.

Method

The technic of postural drainage is as follows: 1. Using an intermittent positive pressure breathing valve, the patient first inhales a nebulized mixture of a bronchodilator and a detergent in 40 per cent oxygen, for the purpose of liquefying accumulated secretions. 2. Then, restrained by an abdominal belt attached to the foot of a specially designed tilt-table, the patient is suspended head first over the edge (fig. 1, *A*), in a position that facilitates drainage by gravity. While in this position, the patient is encouraged to cough, as the assisting therapist taps firmly over the posterior and the lateral aspect of the thorax. 3. At intervals, the patient is turned from one side to the other to aid drainage of pocketed secretions (fig. 1, *B* and *C*). This procedure takes fifteen or twenty minutes. For effective postural drainage, the patient must be put in

such a position that gravity will help the flow of accumulated secretions. This requires a precise knowledge of pulmonary anatomy, as well as an appreciation of proper positioning (fig. 2).

Fig. 2 — Anatomic relations in postural drainage of the bronchi of the right middle and lower lobe.

After maximal drainage and clearance of the bronchial tree are achieved, selected breathing exercises are carried out for the establishment of a pattern of breathing approaching normal. These exercises stress short and abrupt inhalation followed by slow prolonged exhalation; both are regulated by a metronome (two beats for inhalation and three or four beats for exhalation). These exercises are given for five minutes at first, three or four times daily. The following specific exercises are used:

1. The patient reclines in a supine position with the foot of the bed elevated about 20 degrees, and places his left hand over the sternal region and his right hand on the abdomen. He now inhales abruptly through the nose, with the aim of expanding the abdominal wall, and then exhales slowly, while his right hand compresses his abdomen. The average patient has difficulty in mastering the technic of this relatively complex exercise, and ample time should be allowed for him to master it. Then further exercises are added, and the patient changes position from lying down to sitting and to standing, and walks on a level and climbs stairs.

2. When the exercises are mastered, slight resistance is added: With the patient in a sitting and in a standing position he blows out a candle set on a yardstick. As his ability to exhale increases, the candle is moved out along the yardstick.

3. When the maximal distance is attained, additional resistance is provided by an exercise of blowing water from one half-filled half-gallon bottle into another similar bottle connected to the first with rubber tubes.

The exercises are carried out by each patient individually at first and then in a group with other patients. Patients are indoctrinated as to the purpose and importance of the exercises and are urged to practice them at frequent intervals throughout the day in order to achieve maximal improvement in breathing habits.

The value of breathing exercises long has been recognized in the management of patients with pulmonary diseases. Unfortunately, the use of these exercises has met with much skepticism and misgiving, because their effectiveness has thus far not been substantiated by reliable experimental data but has been assessed only empirically, on the reaction of the patients or the subconscious bias of the clinician.

Our group of 81 patients who had undergone the program of exercises just described improved clinically and increased their tolerance to daily activities. However, the improvement could not be measured by routine tests of pulmonary function or by studies of gas in the arterial blood. This led to the impression that the improvement in the pulmonary ventilation of patients with relatively severe impairment is clinically impressive but still insufficient to be recorded by the spirometer. Therefore, other methods for recording respiratory physiologic changes were explored, and an apparently satisfactory answer was found in the measurement of oxygen consumption and determinations of oxygen debt and recovery, described in a previous paper by Gordon and Haas.[11]

The term "oxygen debt" is a convenient expression introduced by Hill and associates[12] to describe the physiologic state following strenuous muscular activities in which the deficit of oxygen intake represents a debt which must be repaid during recovery. In continuous moderate exercise lasting not more than a few minutes, the oxygen available as a rule is adequate to meet the oxygen requirement, and the subject is said to be in a "steady" state. That is, he is able to supply adequate oxygen to keep pace with metabolism. However, if the exercise is so strenuous that the oxygen intake cannot keep pace with the metabolic demand, the subject is no longer in a "steady" state (physiologic equilibrium), and oxygen debt is incurred. This basic concept

applies both to healthy subjects and to patients who have respiratory disorders.

In fact, our observation, supported by studies of oxygen consumption, oxygen debt and recovery, showed that patients from the control group (who had not participated in the rehabilitative measures) were unable to supply adequate oxygen to keep pace with their rate of metabolism after not more than five minutes of light, medium or heavy exercises in daily living. Consequently, these patients were unable to reach the "steady" state, and remained in constant and prolonged oxygen debt, which could be repaid only long after cessation of even slight activity.

Patients with pulmonary emphysema who underwent therapeutic exercises showed definite improvement. In time the tension and respiratory embarrassment with which they performed their exercises were reduced, and consequently there was less oxygen cost. The oxygen debt incurred during the exercises was progressively lessened, as was the period required for recovery. Figures 3 through 6 show average oxygen consumption before, during and after certain specified activities by groups of 10 emphysematous patients who had not done breathing exercises, of the same patients after postural drainage and breathing exercises and of 10 healthy persons.

Our observations showed that although the patients referred to us were

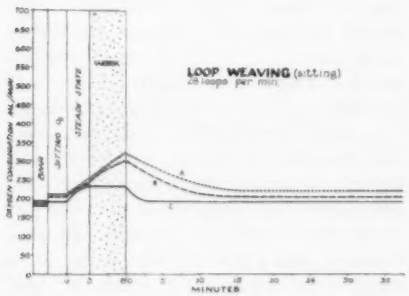

Fig. 3 — Graph showing average oxygen consumption before, during and after loop weaving with patients in sitting position: Line *A*, 10 patients in experimental group before exercise (mean age 58 years, mean body surface area 1.77 square meters); line *B*, 10 patients in experimental group after postural drainage and breathing exercises (mean age 58 years, mean body surface area 1.77 square meters), and line *C*, 10 healthy persons (mean age 55 years, mean body surface area 1.79 square meters).

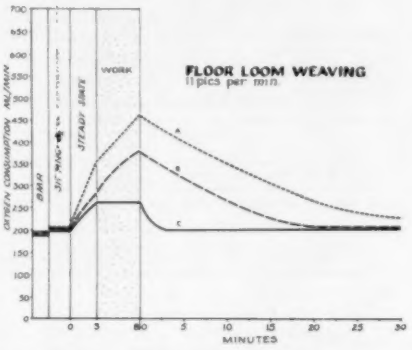

Fig. 4 — Graph showing average oxygen consumption before, during and after floor loom weaving with patients in sitting position: Line *A*, 10 patients in experimental group before exercise (mean age 58 years, mean body surface area 1.77 square meters); line *B*, 10 patients in experimental group after postural drainage and breathing exercises (mean age 58 years, mean body surface area 1.77 square meters), and line *C*, 10 healthy persons (mean age 55 years, mean body surface area 1.79 square meters).

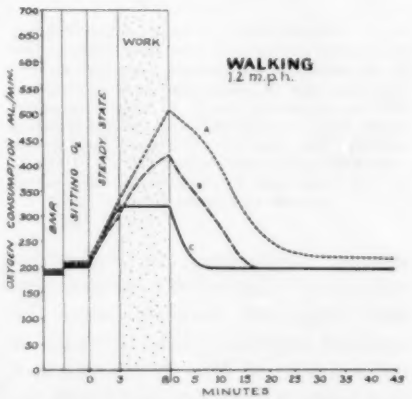

Fig. 5 — Graph showing average oxygen consumption before, during and after walking on level surface: Line *A*, 10 patients in experimental group before exercise (mean age 58 years, mean body surface area 1.77 square meters); line *B*, 10 patients in experimental group after postural drainage and breathing exercises (mean age 58 years, mean body surface area 1.77 square meters); and line *C*, 10 healthy persons (mean age 55 years, mean body surface area 1.79 square meters).

Table 1: Results and Disposition of Experimental and Control Groups

	Experimental Group Hospitalized Patients			Control Group Out-patients		
	Male	Female	Total	Male	Female	Total
Still in hospital	10	2	12	12	0	12
Returned to job; still at work	9	1	10	5	0	5
Awaiting job placement	15	2	17	0	0	0
In workshop training	4	1	5	0	0	0
Rehospitalized	4	0	4	3	0	3
Living in nursing home	2	1	3	8	0	8
Rehospitalized; died	3	1	4*	13	0	13†
Died in nursing home	2	1	3*	0	0	0
Returned to self-care	19	1	20	0	0	0
Lost for miscellaneous reasons	1	2	3	9	0	9
Totals	69	12	81	50	0	50

*Causes of death: pneumonia, 3; congestive heart failure, 4.
†Causes of death: pneumonia, 4; congestive heart failure, 8; cardiovascular infarction, 1.

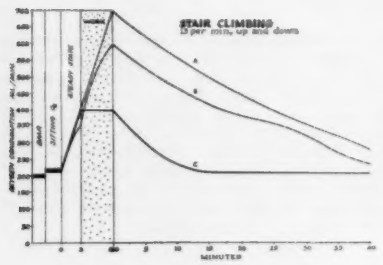

Fig. 6 — Graph showing average oxygen consumption before, during and after stair climbing: Line A, 10 patients in experimental group before exercise (mean age 58 years, mean body surface area 1.77 square meters); line B, 10 patients in experimental group after postural drainage and breathing exercises (mean age 58 years, mean body surface area 1.77 square meters); and line C, 10 healthy persons (mean age 55 years, mean body surface area 1.79 square meters).

severely handicapped by irreversible pulmonary disease, the majority of them improved clinically under the outlined regimen. Their tolerance to the activities of daily living increased to a point where they could care for themselves adequately or completely. Some even were able to return to gainful work. The retraining for the latter was more successful with the experimental group than with the control group. Table 1 shows the results and disposition of the two groups.

Summary

There is a relative lack of utilization of rehabilitative measures in the treatment of patients with pulmonary emphysema. To evaluate the effectiveness of these measures, a study was initiated by the Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center. This study is now in the fourth year. Eighty-one patients have been hospitalized at Goldwater Memorial Hospital to date. These patients had been frequently hospitalized previously, and forced idleness had followed, since they were unable to retain their employment. They had been previously treated, primarily, for symptomatic relief, with antibiotics, bronchodilators and oxygen. Although these remedies served to lessen discomfort, they did not affect the progress of the disease, since they could not change the pulmonary abnormality, which is, as a rule, irreversible. Our study showed that physical rehabilitation of emphysematous patients by means of postural drainage combined with therapeutic breathing exercises resulted in unquestionable improvement, in comparison with patients who had not undergone these remedial measures.

There is prevalent a general skepticism as to the value of these procedures,

possibly since their effectiveness has thus far not been scientifically demonstrated. Spirometric evaluation has been attempted, but this measure definitely lacks the sensitivity desirable for detecting any significant change in pulmonary function. Further investigation into this problem disclosed that improvement in pulmonary function after respiratory exercises could be readily determined by measurement of energy cost, oxygen debt and recovery rate.

Conclusion

1. Impairment of pulmonary ventilation definitely can be lessened with the aid of postural drainage combined with breathing exercises.

2. These therapeutic modalities have a definite place in the over-all treatment of patients suffering from chronic pulmonary emphysema.

3. These measures should be carried out with perseverance, with the objective of correcting, if possible, the patient's impaired breathing pattern and teaching him to utilize economically his limited cardiopulmonary reserve.

4. Improvement in pulmonary function should be determined at intervals by measurement of energy cost, oxygen debt and recovery.

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Local Injection of Corticosteroids in Treatment of Musculoligamentous Injury

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• This paper reports the technic and results of local infiltration of soluble prednisolone phosphate used alone or in combination with other steroids and local anesthetics in a wide variety of soft tissue injuries. Rapid reduction of swelling and alleviation of pain are prime objectives for earlier rehabilitation. The soluble corticosteroid is more rapid, acting with maximum effectiveness usually within 24 hours. The less soluble suspension, such as prednisolone tertiary-butylacetate, contributes a more sustained anti-inflammatory and fibrinolytic effect. The technic of "flooding" large areas is described, as well as that of combining corticosteroids with hyaluronidase, where ecchymosis or edema are prominent features. Typical case reports, including collateral ligament strain, epicondylitis, cervical strain, low-back strain, ankle strain and bicipital tendinitis, are included.

The injection of corticosteroids in collagen into the joints and soft tissues, and in vascular diseases has been well documented. Recently, however, the results of local infiltration of steroids in the treatment of injuries to soft tissue have been favorably reported [1, 2, 3, 4, 5]. Beneficial effects from the local infiltration of hydrocortisone, crystalline suspension of prednisolone acetate and water-soluble prednisolone sodium succinate solution have been reported. The conditions treated were usually those following athletic injuries involving the soft tissues.

This paper reports the technic and result of local infiltration of soluble prednisolone phosphate, used alone or in combination with other steroids and local anesthetics, in a wide variety of injuries to soft tissue. Reports of typical cases are included. More than 1,200 private patients were treated during almost two years. Private patients deliberately were selected for evaluation of the results, because subjective symptoms play an important role, and adequate follow-up is required. Conditions treated varied from simple ligamentous strain to multiple injuries to soft tissue associated with serious skeletal fractures.

Of prime importance in the treatment of injuries are the reduction of swelling and alleviation of pain. The intramuscular and extra-articular application of a steroid suspension plus flooding with soluble corticosteroid (with or without an anesthetic) can be employed to advantage in the reduction of inflammatory reaction and the elimination of pain associated with trauma.

In our series, a local anesthetic* was employed for immediate relief of pain. This may be mixed in the syringe with a fast acting soluble corticosteroid, such as prednisolone 21-phosphate**, to achieve rapid anti-inflammatory action. In the same syringe a long-acting corticosteroid, such as hydrocortisone or prednisolone suspension, can be employed for a sustained anti-inflammatory and fibrinolytic effect. When edema or ecchymosis is a prominent feature with trauma to soft tissue, hyaluronidase*** was added to the mixture in the syringe. It is to be emphasized that in all cases reported, supportive treatment, including analgesic medication, physical therapy and pertinent orthopedic procedures, was used, as well as the local injection of corticosteroids.

Preliminary studies were carried out on white rats, in which intramuscular and extra-articular injections of prednisolone 21-phosphate and prednisolone tertiary-butylacetate**** were made in soft tissues which had been subjected to controlled trauma. The animals were killed at varying intervals after trauma, and gross and microscopic examination made. In no instance did we observe

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* "CYCLAINE" (Hexylcaine Hydrochloride 1% Solution) Merck, Sharpe and Dohme Research Laboratories, West Point, Pa.

** "HYDELTRASOL" (Prednisolone 21 Phosphate, 20 mg./cc.) Merck, Sharpe and Dohme Research Laboratories, West Point, Pa.

*** "WYADASE" (1500 USP TRU per cc.) Wyeth Laboratories, Philadelphia, Pa.

**** "HYDELTRA"-T.B.A. (Prednisolone-Tertiary Butylacetate, 20 mg./cc.) Merck, Sharpe and Dohme Research Laboratories, West Point, Pa.

Read at the 3rd International Congress of Physical Medicine, Session on Arthritis, Washington, D. C., August 23, 1960.

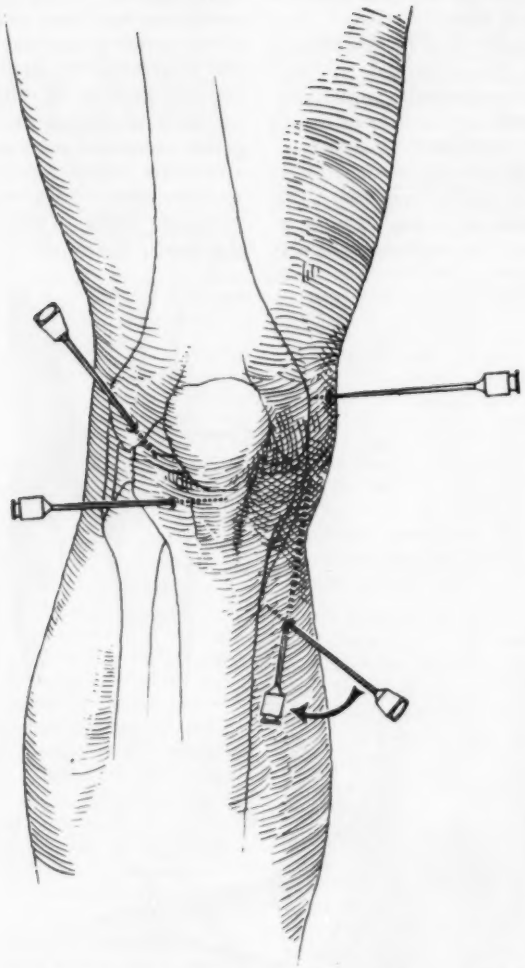

Fig. 1 — Placement of needles for medial collateral ligament sprains; placement of needles for injection of patellar ligament also is shown.

any increase in edema, congestion, accumulation of leukocytes or liquefaction in the treated tissues as compared with the traumatized untreated controlled tissues. Local toxic manifestations did not occur in the tissues examined. This result was consistent with our clinical impression: We have not yet observed a single incident of local or systemic toxic effect.

Our good results and the absence of infection may be attributed in large part to meticulous attention in preparation of skin and sterile technics in all injections into soft tissue. Such precautions cannot be overemphasized. Needles should be autoclaved daily, appropriate needle gage and length selected and site of injection clearly delineated and adequately prepared. Since more than one solution often is mixed in the syringe, it is strongly recommended that the physician himself prepare each injection immediately before he administers it.

In our experience many varieties of acute and chronic trauma to soft tissue were treated. A partial list of these conditions includes: strains and sprains of all major joints and small joints of the extremities; muscle tears and contusions; injuries to tendons; tears and avulsion of ligaments; soft tissue injuries associated with fractures, before and after reduction. The following representative cases suggest the appropriate office management of some commonly encountered conditions.

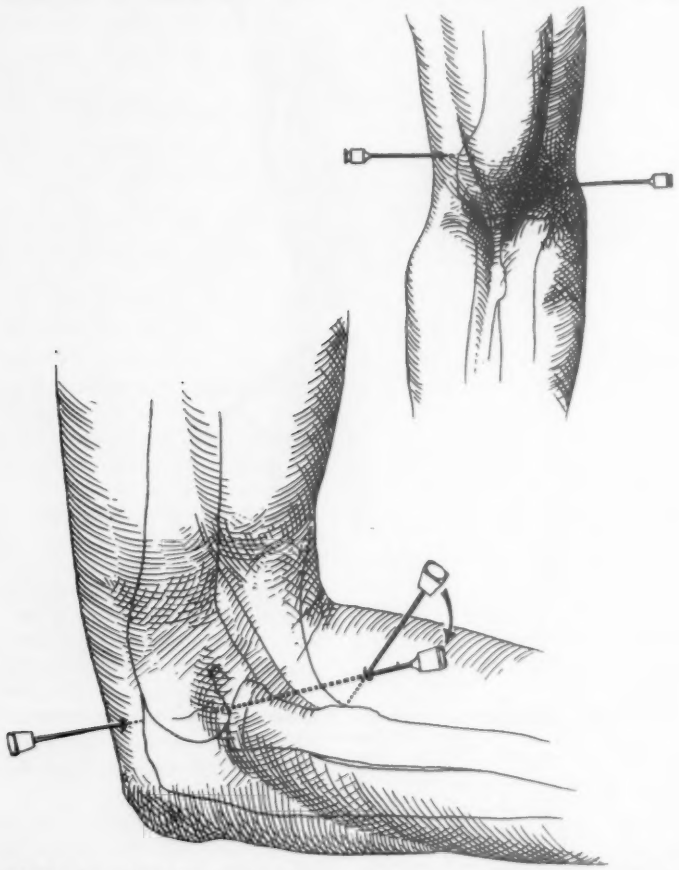

Fig. 2 — Injection for epicondylitis showing placement of needles in the bicipital tuberosity of the radius as well as the epicondyle.

Report of Cases

Case 1.—Medial collateral ligament sprain (fig. 1): A 65-year-old nun (Sr. G.) was first examined for pain in the right knee of two weeks' duration. Pain increased when she ascended or descended steps or genuflected. Roentgen study of the knee showed no essential abnormality. There was considerable tenderness of the medial collateral ligament of the right knee, particularly at its tibial insertion. A diagnosis of sprain of the medial collateral ligament was made. The patient was treated by injection the area of the ligament and its insertion being flooded with prednisolone phosphate (20 mg.) and prednisolone tertiary-butylacetate (30 mg.) mixed with 1 per cent solution of hexylcaine hydrochloride (1 cc.) and sterile physiologic solution of sodium chloride. A compression bandage was applied. Immediate improvement was noted, and the patient continued with her duties. Seven days later she reported some residual discomfort in the same area and was treated in a similar manner. Three days after the second injection, the patient reported that she was free from pain. She remained free from symptoms.

Case 2.—Epicondylitis (fig. 2): A 40-year-old white woman (Mrs. F. M.) complained of severe pain in the left elbow of several months' duration. Pain was most severe when she lifted her child, wrung clothes or sewed by hand. She was unable to use her arm for lifting when the forearm was in a pronated position.

Examination showed marked tenderness in the lateral condyle of the left humerus and over the bicipital tuberosity of the left radius. An injection of prednisolone 21-phosphate (20 mg.) and 1 per cent solution of hexylcaine hydrochloride (1 cc.) was made in the region of the bicipital tuberosity of the left radius and the lateral condyle of the left humerus. A compression bandage was applied and the patient advised to use heat locally. There was almost immediate alleviation of symptoms. Five days later, however, the patient reported that the pain had gone from the region of the tuberosity but persisted in the region of the left lateral condyle. A second injection of prednisolone phosphate and hexylcaine hydrochloride was given in this region. A week later the patient reported some improvement, but pain still was present in the area of the condyle. A third injection, in which prednisolone phosphate was combined with 15 mg. of prednisolone tertiary-butylacetate and hexylcaine hydrochloride, was given in the region of the lateral condyle of the left elbow. Three days later the pain had completely disappeared, and the patient remained free from symptoms.

Case 3.—Severe Cervical Strain (fig. 3): A 44-year-old white woman (Mrs. J. B.) sustained a snapping injury to her neck in an automobile accident. The patient was first examined by one of us two weeks after conservative treatment by her family physician. She was in a state of high emotional anxiety, held her head and neck rigidly and complained of severe pain in the back of the neck and radiating to both shoulders. She experienced occasional paresthesia in both arms also.

Examination showed considerable spasm of both trapezii and some tenderness to deep palpation around the left lateral aspect of the neck. There was considerable pain associated with all ranges of motion of the cervical spine. Diagnosis was severe cervical strain, secondary to the accident. Treatment consisted of infiltrating injections into the groups of spastic muscles in the paravertebral region of 30 mg. prednisolone phosphate and 20 mg. prednisolone tertiary-butylacetate in hexylcaine hydrochloride and sterile solution of sodium chloride. Muscle relaxants and analgesics, which had been prescribed previously, were continued. Cervical traction also was done immediately after the injection. Two days later she reported that she was considerably improved, but still had some pain and tenderness in the posterior cervical region. Sixteen days later she reported that she was much improved, and had only occasional stiffness of the neck. Five months later the patient had a recurrence of similar pain in the shoulders and neck. She was treated by paravertebral infiltration of these muscle groups, with the same preparation used before. This was followed by immediate improvement, and there was no subsequent recurrence.

Case 4—Recurrent Strain of the Lower Part of the Back (fig. 4): A 39-year-old white woman (Mrs. E. DuR.) had had recurrent pain in the lower part of the back for six weeks. Approximately one week before examination she had slipped in the wet grass and the pain in the back became acute. She had a fifteen year history of recurrent low back pain, treated and untreated.

Examination showed spasm of the right erector spinae muscle from L-1 to L-5, with a point of maximum tenderness to the right of L-5 and S-1. The Laguerre and Lasègue leg tests gave positive results on the right and negative results on the left. Otherwise the examination was not remarkable. Neurologic examination showed no abnormality. The legs were of equal length. Forward, backward and left lateral bending were painful, and pain was referred to the area previously described. Roentgen study showed a congenital anomaly consisting of fusion of the fifth lumbar vertebra at the neural arch. A diagnosis of recurrent traumatic strain of the lower part of

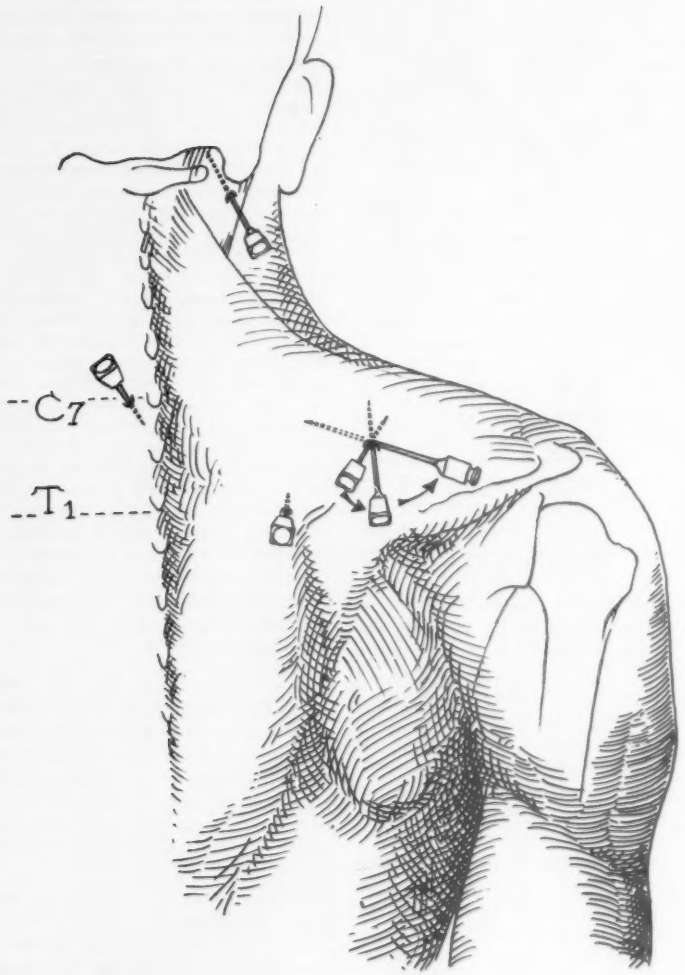

Fig. 3—Injection into the interspinous ligament between C-7 and C-8 as well as infiltration of the suboccipital area and trigger points in the trapezius muscle and upper angle of the scapula for cervical strain.

the back was made.

Treatment consisted of paravertebral block and infiltration of the spastic muscle to the right of L-1 through L-5. A 20 cc. mixture of 3 cc. (60 mg.) prednisolone tertiary-butylacetate, 2 cc. (40 mg.) prednisolone phosphate, 5 cc. 1 per cent solution of hexylcaine hydrochloride in sterile solution of sodium chloride was injected. The patient reported some improvement, but five days later some symptoms associated with muscle spasm in the same area persisted. The procedure was repeated. Two days after the second injection the patient reported disappearance of pain, but there was residual soreness. Local heat and other conservative means of treatment were used to treat the residual soreness, and the patient made a prompt and uneventful recovery. There had been no symptoms for two years at the time this report was made.

Case 5.—Chronic Sprain of the Ankle (fig. 5): A 28-year-old woman (Mrs. E. H.) was first examined on June 9 because of limping and pain in the left ankle. She stated that in February, after she slipped in the snow, her ankle had twisted and she began to limp. Pain, swelling and limp persisted despite repeated efforts at treatment, including immobilization in a cast for three weeks, together with other forms of conservative management. Roentgen study showed no abnormality of bone.

Examination showed sprains of the left talofibular ligaments, calcaneofibular ligament and peroneal tendon. A diagnosis of severe chronic sprain of the ankle was made.

Treatment consisted of an injection of hyaluronidase, 30 mg. prednisolone tertiary-butylacetate, 40 mg. prednisolone phosphate and solution of hexylcaine hydrochloride to flood the region of the talofibular and calcaneofibular ligaments. This was followed by local application of heat, strapping of the ankle and prescription of shoes with longitudinal counters. The patient reported immediate relief. One week later she was reexamined. Pain was minimal, and a new strapping was applied. A few months later the patient reported that she was free from symptoms, with no pain or limp. She had continued to wear her prescribed shoes, but no further treatment was required.

Case 6.—Chronic Traumatic Bicipital Tendinitis: A 48-year-old white woman (Mrs. H.) complained of pain in the right shoulder. She stated that thirteen years before examination a fall on her outstretched arm resulted in severe pain in the shoulder. She was treated for "bursitis" by roentgen irradiation of the right shoulder. There was improvement from the acute pain, but from that time to the time of examination she had had recurrent nagging pain in the region of the right shoulder. Pain had been present also on abduction and forward flexion of the arm throughout the thirteen years, with acute exacerbations at various times.

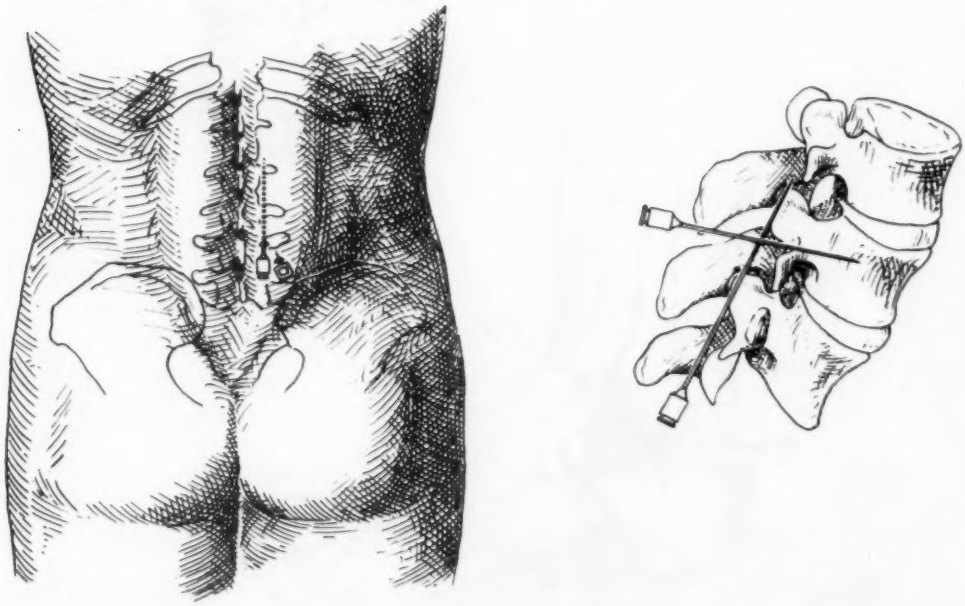

Fig. 4 — Technic for injection of back strain utilizing infiltration of painful muscles and the paravertebral area.

Examination showed tenderness over the anterior aspect of the right shoulder in the region of the bicipital groove. Considerable tenderness simulating the pain during exacerbations was associated with rolling of the bicipital tendon under the examiner's finger. Abduction of the shoulder was not restricted, but pain was present at about 135 degrees. On forward flexion, pain was present at approximately 90 degrees. Roentgen study showed an essentially normal shoulder. A diagnosis of chronic traumatic bicipital tendinitis was made.

The patient was treated by means of three injections at five day intervals. An injection of 30 mg. prednisolone phosphate and 30 mg. prednisolone tertiary-butylacetate, with 1 cc. of 1 per cent solution of hexylcaine hydrochloride and hyaluronidase was given, in a 3½ inch (8.89 cm.) needle. The area of the bicipital tendon and the intertubercular sulcus was flooded, the needle being inserted in the direction of the long axis of the tendon. The patient reported immediate improvement after the first injection, and was able to increase motion without pain. After the third injection she remained free from pain and enjoyed unlimited range of motion.

Comment

Before any consideration is given to the use of corticosteroids by injection in treatment of injuries to soft tissue, a correct diagnosis must be firmly established. Frequently the so-called simple sprained ankle is found to be a fracture of the malleolus, which may necessitate different treatment. Adequate roentgen examination is necessary not only for positive diagnosis, but to rule out skeletal lesions.

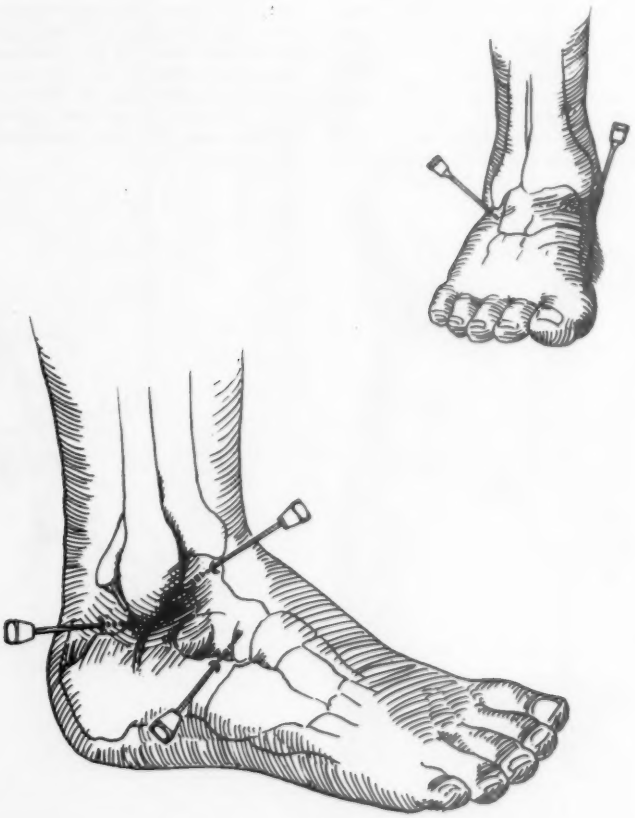

Fig. 5 — Infiltration of extra-articular areas for ligamentous strains about the ankle.

SUMMARY OF RESULTS OF TREATMENT

Total number of patients treated 200
Total number of conditions treated 205
Number of injections used 405

Location of conditions treated			Total No. injections	Average No. injections	Results: Excellent	Good	Poor	Fair	Unknown
Cervical spine:									
Cervical strain	5								
Cervical syndrome	14								
Cervical sprain	2								
Cervical myositis	5								
TOTAL	26		46	1.7	13	8	1	2	2
Dorsolumbar spine									
Lumbosacral sprain	6								
Lumbar strain	15	(acute, 9; chronic, 6)							
Thoraco-lumbar strain	7								
Sacrococcygeal strain	1								
Sacroiliac strain	1								
TOTAL	30		60	2	19	7	2	0	2
Shoulder girdle									
Calcareous tendinitis	6								
Subacromial, subdeltoid bursitis	9								
Bicipital tendinitis	9								
Supraspinatus strain	1								
Myositis	1								
TOTAL	26		48	1.7	22	3	0	1	1
Elbow									
Epicondylitis	6								
Forearm muscle strain	2								
TOTAL	8		21	2.6	5	2	0	1	0
Hip									
Sciatic Syndrome (soft tissue injections)	2								
Tensor fasciae latae—trochanteric syndrome	13								
Adductor strain	3								
Rotator strain	2								
TOTAL	20		44	2.2	15	2	3	0	0
Knee									
Med. Collat. Lig. strain or sprain	12								
Lat. Collat. Lig. strain or sprain	4								
Hamstring insertion strain	2								
Patellar tendinitis	4								
Parapatellar contusions	2								
Periarthritic inflammation	2								
TOTAL	26		61	2.3	18	4	0	2	2
Ankle									
Strain	3								
Sprain	7								
Contusion	3								
Chronic sprain	1								
TOTAL	14		22	1.4	11	2	0	0	1
Trigger point									
In scapula and ribs	13								
Intercostal	1								
TOTAL	14		30	2.1	11	2	0	0	1
Miscellaneous (includes injuries to muscle and all types of injuries to soft tissue, ranging from simple contusions, with attending myositis, to severe sprains, of fingers, toes, wrists, forepart of foot, metatarsus and metacarpus).									
TOTAL	40		73	1.8	27	4	4	1	4
TOTAL	405		405	2.0	141	34	10	7	13

In all cases, either prednisolone phosphate, prednisolone tertiary-butylacetate or hydrocortisone tertiary-butylacetate, together with 1 per cent hexylcaine hydrochloride or xylocaine, was used; in conjunction with hyaluronidase when ecchymosis, edema or both were present.

The relatively high number of unknown end-results is due to the fact that the patients were treated in private practice, and apparently felt no need to return for a final examination. It is only to be assumed that a fair number of these were satisfied with the results.

Individualization of treatment is necessary because of the wide variation in trauma and its effect on various patients. The amount of local anesthetic, corticosteroid or hyaluronidase used will vary with the severity and extent of the injury. When large areas of soft tissue are involved, the active medicament may be dispersed with increased effectiveness if it is diluted with sterile physiologic solution of sodium chloride. Careful questioning regarding sensitivity to the "caine" drugs should precede injection, to prevent injudicious use of a local anesthetic for a sensitive person. The dose of prednisolone phosphate varied from 10 mg. to 40 mg. When prednisolone tertiary-butylacetate was employed, the dose ranged from 5 mg. to 60 mg.

Care must be observed in the proper placement of the needle during injection. The plunger of the syringe should be withdrawn before each injection, to avoid penetration of a blood vessel. In ligamentous or tendinous areas, caution should be exercised to avoid injection directly within the tendon or ligament. When the needle is properly placed, the medicament can be injected gently, without encountering undue resistance. In many instances it is possible to "flood" an area with the medicament rather than concentrate the entire contents of the syringe in a small area. If severe pain is experienced when the needle is inserted, it should be withdrawn to avoid injury to a nerve. "Trigger" points, often found as points of maximum tenderness in the presence of traumatic myositis, may be used as sites for injection. Specific points of injection for traumatic lesions depend on the correct diagnosis. Injections into the appropriate ligaments, tendons and lesions of soft tissue should be done on an individual basis. If a local anesthetic is employed, the patient must be forewarned that only temporary immediate analgesia is expected. It is encouraging, however, that we have experienced few flare reactions after the effect of the anesthetic has worn off. In our experience, other corticosteroids in soft tissue injections may be more irritating than prednisolone phosphate after the local anesthetic effect has worn off. Some subjective relief is experienced within a few hours when the preparations and methods described herein are used. When only soluble prednisolone phosphate is used, maximum relief usually is noted within twenty-four hours. When the insoluble corticosteroids are added to this, relief will last longer. A single injection may be sufficient when trauma is not extensive; after severe injury or when treatment has been delayed, repeated injections may be required at five day intervals until the desired result is obtained.

We consider local injection of corticosteroids only an adjunct to sound orthopedic and physical medical management. Perhaps its chief value is not so much as a cure in itself as in permitting early rehabilitation and speeding recovery. Our aim in treatment has been to relieve symptoms rapidly and enable the patient to resume normal activities. It has been our observation that physical medical procedures, such as exercise to maintain joint range and muscle strength, were begun sooner and carried out more effectively when corticosteroid medication had been done, and that swelling and edema were reduced more efficiently than when this treatment was not used.

In conclusion, our experience has shown that the use of a combination of a soluble corticosteroid and a suspension of a longer-acting steroid is effective in treatment of injury to soft tissue. The addition of local anesthetics, hyaluronidase and physiologic solution of sodium chloride speeds the elimination of pain and causes a decrease of edema, so that dispersion of the active anti-inflammatory steroids is improved.

Summary

The technic and results of local infiltration of soluble prednisolone phosphate, used alone or in combination with other steroids and local anesthetics in the treatment of a wide variety of injuries to soft tissue, are reported.

Rapid reduction of swelling and alleviation of pain are prime objectives, to lessen the time required for rehabilitation. The soluble corticosteroid works more rapidly than less soluble suspensions, such as prednisolone tertiary-butylacetate. The soluble preparation reaches maximum effectiveness usually within twenty-four hours, while the suspension contributes a more sustained anti-inflammatory and fibrinolytic effect.

The technic of "flooding" large areas is described, as well as the method of combining corticosteroids with hyaluronidase when ecchymosis or edema is a prominent feature. Reports of typical cases, including instances of strain of a collateral ligament, epicondylitis, cervical strain, strain of the lower part of the back, ankle strain and bicipital tendinitis, are included.

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Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.

special article

Natural History of the Intervertebral Disc

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Chicago

● Associated with growth and maturation is denaturation of the collagen and mucopolysaccharides of the intervertebral disc. Denaturation changes are ubiquitous and inevitable, although the stage may vary from person to person of the same age and even from disc to disc in the same person. This process in the nucleus pulposus leads to loss of elasticity and, consequently, interference with radial dispersion of forces. Simultaneously, aging in the annulus fibrosus produces a fibrous, rigid structure which becomes the seat of fissures and frank tears due to constant thrust from within by a nucleus less efficient in transmitting forces equally in all directions and so incapable of minimizing them in any sector of weakened resistance. This background of dynamic processes affecting the sensitive internal milieu that comprises the intervertebral disc may offer a rationalistic approach to a multiplicity of clinical syndromes involving the vertebral column. Certain conclusions may then be postulated regarding the diverse nature of "discogenic" pain commonly seen in the neck and low back regions.

Implicit in the title is progressive change of the intervertebral disc and, consequently, the vital concept of aging. This term must be redefined for the purposes of the discussion. In clinical understanding aging is a temporal concept expressing the association of disease with later life. Biologically speaking, however, aging refers to a cycle of growth coexistent with the life history of the individual and characterized by maturation and then decline, not necessarily associated with but often leading to disease. This definition of aging is the primary one and describes all living tissue. The following discussion will, therefore, be concerned with the cycle of aging, as it alters the anatomic, physicochemical, biochemical and functional properties of the intervertebral disc, and with associated events manifested during life. A synthesis will then be attempted between postmortem, experimental, and clinical observations, in order to rationalize syndromes involving the vertebral column, particularly the lumbar and cervical portions. In more familiar terms, I refer to mechanisms of common back and neck pain. The report does not concern vertebral fractures and infections, rheumatoid spondylitis, neoplasms and other diseases of the bone proper.

Since all living tissue undergoes the biologic process of aging, any description of a disc taken from a single point in time will have serious limitations. Keyes and Compere[1] have summed up this difficulty concisely: "No normal (disc) can be accurately described unless the age be specified." Consequently, the following section relates to properties derived from studies of a "normal" disc, that is to say, from humans early in life, or from mammals of an analogous stage of development. For it may be stated at the outset that in the mammalian world man is not unique in his back pain; he is only more articulate. With minor variations intervertebral disc syndromes have been found in cattle, cats and, commonly, the dachshund, spaniel, pe-

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kingese and French bulldog.[2] Obviously, if factors of stress and strain contribute to such derangements, they are operative regardless of the posture of the animal.

Anatomy of the Intervertebral Disc

In a cross-section of this structure the central nucleus pulposus stands out sharply homogeneous as a glistening, gelatinous, elastic globoid. It is enclosed in a denser, firm, white envelope, the fibrocartilaginous annulus fibrosus. Newer methods recently have considerably extended the view obtained from the ordinary light microscope. With this tool only the general plan of all connective tissue can be appreciated: fibrils embedded in a uniform ground substance. But what by ordinary microscopy appear as a few faint "reticular" strands in the nucleus pulposus are now revealed in the electron microscope as a rich network of fine banded fibrils (width 50 $m\mu$), lying in randomly oriented directions and uniformly dispersed in the homogeneous matrix.[3] In the annulus the elastic mechanism is quite different. Although the fibrocartilage has been known to consist of obliquely disposed fibers arranged in layers, the polarized light microscope and x-ray crystallography reveal that the fibers have a biaxial arrangement, so that one sheet of fibers interlocks at an angle of 60 degrees with each adjacent sheet, forming the so-called "interstriation angle."[4] This arrangement imparts considerable adaptation to deforming forces.

Biochemistry

On the molecular level two types of substances appear whose natures, though distinct, are related by virtue of the fact that they are both macromolecules. These substances are collagen and mucopolysaccharides; together with fibroblasts all three comprise the basic constituents of connective tissue.

Macromolecules are built up from polymerization of smaller, simpler molecules. In the case of collagen the units are 20 different amino-acids. The molecule is in the form of three long chains in spiral arrangement around a common axis and has a molecular weight of 350,000; as derived from x-ray diffraction studies, it is 300 $m\mu$ long and 1.35 $m\mu$ wide.[5] Though not as well defined as collagen, another protein occurs which is intimately associated with the mucopolysaccharide making up the homogeneous matrix mentioned above. This association is referred to as mucoprotein having the enormous molecular weight of four million, and is regarded as a loose chemical binding between the protein and a polysaccharide polymer.

The latter is the second class of macromolecule referred to. It presents an entirely different composition from protein, as its basic polymeric pattern is a repetition of two linked derivatives of a hexose sugar. One derivative is a hexose acid (hexuronic acid), and the other an N-acetylhexosamine or, in other words, an acetylated amino-sugar. These two simpler units form an acid disaccharide which repeats itself "n" times in the complete polymer. In addition, a disaccharide may possess a sulfate radical linked to the hexosamine moiety. Another chemical variation in the basic disaccharide structure is that of stoichiometric isomers. The hexuronic acid presents itself in one of two forms, either glucuronic acid (an analogue of glucose) or its isomer, iduronic acid. Similarly, either N-acetylglucosamine or its isomer, N-acetylgalactosamine, may constitute the other portion of the disaccharide unit.

As examples of common polysaccharides these may be mentioned: hyaluronic acid, occurring in vitreous humour and joint fluid, which is a combination of glucuronic acid and N-acetylglucosamine; chondroitin sulfate C, plentiful in nucleus pulposus, a sulfate ester of galactosamine combined with glucuronic acid. An exception to the presence of a hexuronic acid in the disaccharide unit is known. A polysaccharide called keratosulfate is built up of sulfate-acetylglucosamine

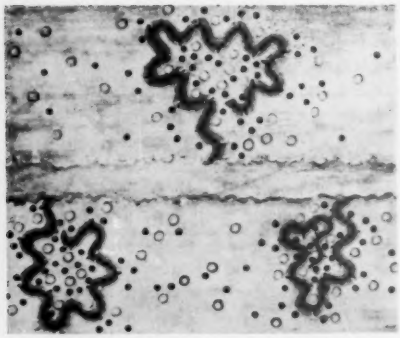

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Fig. 1 — Schema of a part of the protein-mucopolysaccharide complex. The center core represents the protein. The wavy black lines represent the polysaccharide chain. The black dots are sodium ions; the circles are chloride ions. The gray background above and below the protein core represents water of the milieu.[5]

and the sugar galactose (instead of a hexose acid); the resulting mucopolysaccharide appears to be abnormal as will be discussed in a following section. Although found normally in small amounts, it occurs plentifully in connective tissue from those with Marfan's syndrome as well as in intervertebral discs late in life.

The presence in the ground substance of macromolecules rich in negative acid radicals (—COOH and $-SO_2OH$) endows upon the complex a strong cation combining capacity. Since ions tend to bind water, hydration of the ground substance becomes understandable. In this light, the extracellular fluid can not be regarded as being held in connective tissue as water in a sponge.

In the ground substance, then, a protein (non-collagen) is combined with long chains of mucopolysaccharides. Figure 1, adapted from Dorfman, schematizes the essential structure of such a mucoprotein.[5] It has been established that the ratio of protein to polysaccharide is one to four by weight and that, further, metabolic turnover in the cartilage of rib, for example, is equal for both, as would be essential for the integrity of the complex. The collagen molecules have a slower turnover in young tissue. With aging this becomes so reduced that replacement probably plays little or no part in repair of the intervertebral disc.

Physical Chemistry

The nucleus pulposus represents a colloid solution of the gel type in which the fine, randomly oriented fibrils of collagen are homogeneously dispersed throughout an abundant hydrophilic ground substance (protein-mucopolysaccharide complex). This is another way of stating that the protein is in a soluble state. It may be extracted with saline or citrate to form another well known gel, viz., gelatine. It may also be precipitated from a solution into fiber formation by changes in pH, temperature, and salt concentration. In the annulus the collagen fibers are more numerous and heavier; but they also appear to be in dispersed state consistent with a gel. We have seen that the elasticity of the annulus is largely accounted for by its mechanical arrangement of fibers to form the interstriation angle. Of interest and extreme importance to further development of the subject, we must further examine the basis of the remarkable versatility displayed by the nucleus pulposus both in strength and elasticity.

The nucleus possesses a hydration pressure because of its gel state. Although it has been assumed that the pressure has an osmotic basis, Hendry convincingly marshals proof that this can not be the cause.[6] Rather, hydration gives rise to an "imbibition pressure". This depends on the "hydrophilic", or water-attracting, nature of the nucleus pulposus with its colloid-mucoprotein composition and the large number of anionic radicals which attract cations along with water of hydration. The resulting affinity for water is responsible for the imbibition pressure. This may be enormous as seen, for example, by the fact that swollen peas can split rocks. It accounts for the functional adaptation of the nucleus pulposus whereby the imbibition pressure balances the external pressure upon the vertebral column.

Function

The disc, aside from imparting mere support in common with connective tissue, is admirably built for buffering forces imparted to the vertebral column. Because its structure confers a high degree of elasticity upon the nucleus, it acts as a hydraulic cushion radially dissipating end-to-end forces uniformly to the annulus. The latter accommodates itself to the outward thrust by virtue of the interstriation angle and tends to flatten slightly. A critical physicochemical equilibrium between colloid and tissue fluid is maintained by the protein-mucopolysaccharide structure. This reactive mucoid substance is sensitive to changes in its physical and chemical environment. What they are is unknown, but it is clear that if these changes were better understood, the process of aging and consequent altered function of the disc and, indeed, of all connective tissue may be predicated upon them.

Aging or Denaturation of the Disc

In discs of older animals, differentiation between annulus and nucleus becomes less and less distinct. Grossly, the latter becomes dry, firm, even crumbly; it loses its glistening, mucoid appearance and comes to resemble the outer fibrocartilage. By the ordinary miscroscope, an increase and coarsening of fibrils are apparent at the expense of the mucoid ground substance.[1] Electron microscopy confirms the general change; collagen is aggregated into dense fibrils and the ground substance appears clumped.[3] The polarizing microscope, furthermore, reveals the loss of the biaxial arrangement of fibers in the annulus, so that the interstriation angle disappears.[2] In addition Schmorl reports from autopsy material that calcification is a feature in 71 per cent of annuli and seven per cent of nuclei.[7]

The most striking and illuminating evidence of aging of the disc comes from physicochemical and biochemical considerations. In general, alterations in the nature of the collagen and the mucoprotein (the protein-polysaccharide complex) are consistently present in discs from aging mammals. These phenomena are similar to those produced in the laboratory and represent molecular rearrangements common to that class of substances that fall under the heading of macromolecules.

During the life cycle the colloidal solution of collagen passes from the gel to an insoluble state. At the outset fine and randomly oriented, the fibers crystallize into large, coarse aggregations directed *in one plane*. In fact, this physicochemical description of collagen fibers in the aging disc corresponds to those denatured by heat. Secondly, along with the loss of the gel state, a progressive decrease in the relative water content of both annulus and nucleus is well documented.[1,7] Table 1 demonstrates the percentile fall in water content of the disc. This relative decrease is also absolute, as there is evidence of reduction in volume of the intervertebral disc. A third alteration is found in the variations of intradisc pressures, but here agreement ceases. As reported by Schmorl in his large autopsy material, up to 40 years of age it rises and then falls in subsequent decades as the disc shows well marked degeneration.[7]

Table 1: Decline in Water Content with Age in Nucleus and Annulus of Disc

Age	N.P.		A.F.**
	%*	%**	%
Birth	88	88	76
12	80	—	
20's	—	76	70
70's	70	70	70

*Keyes and Compere[1]
**Schmorl and Junghanns[7]

On the other hand, on grounds of experimental error, Hendry denies the increase of intradisc pressure in midlife.[6] He demonstrates a progressive loss of hydrophilia and hydration, due to a more labile water-binding capacity. Thus while external pressures force only a minimal quantity of fluid from the young nucleus pulposus, those undergoing alterations show relatively large losses in water content when

equal forces are applied. During life in the latter instance external pressure could produce an acute fall in volume as water of imbibition is forced out. Only when the nucleus is finally reduced to a coarse fibrous structure of low hydrophilia is its volume stabilized. Hendry is convinced that mechanical derangements of the disc stem from loss of elasticity which in turn results from reduced water-binding capacity.

The biochemical findings in aging center about the polysaccharides.[5] The ground substance yields a progressively smaller concentration of these macromolecules, reflected in a diminishing polysaccharide : collagen ratio. Evidence of progressive depolymerization of the former substance is striking, accounting at least in part for this altered ratio. The initially small amount of keratosulfate in the nucleus pulposus in later decades increases at the expense of true acid mucopolysaccharides, so that the keratosulfate: chondroitin sulfate ratio increases. Accordingly, there is not only a reduction of mucopolysaccharides, but functionally poor ones replace normally reactive molecules. This abnormal composition interferes with the water-binding action of the ground substance. In addition, the polysaccharides appear to bind preferentially the denatured colloid, as is strikingly demonstrated in electron microscopy,[3] a further indication of degraded function.

It is apparent that altered physicochemical and biochemical properties and composition of the disc account for the different anatomic appearance seen in older animals. What is more important, they probably underlie changes in function. First, loss of the gel state, as well as reduction in water-binding capacity of acid mucopolysaccharides of the nucleus, disturbs its elasticity which normally allows equal and radial dispersion of deforming stresses. Along with shrinkage, impaired elasticity produces unequal distribution of forces. Thus, a somewhat more rigid section of the nucleus may act like a battering ram against the annulus fibrosus, which itself is now becoming ill-adapted to receive such blows, thanks to the loss of the interstriation angle of the fibers and change in its colloidal properties. If Hendry is correct the alternate fluctuations in nuclear volume with loading and unloading add further to the accumulative trauma to the annulus.[6] The constant battering against rigid, unelastic tissue leads to faults, tears and defects in the annulus. During the years the nucleus pulposus gradually shifts from a central to a posterior position. Added to this sequence of events is the significant laboratory finding that the aging disc requires a lesser intensity of trauma necessary to produce rents.[5] Perhaps, such time-linked occurrences help explain the high incidence of low back pain and mechanical derangements in the later decades.

Granted the aging of the macromolecules, the question may be raised whether new collagen can counteract the process by replacing the defective protein through metabolic turnover. This expectation is probably not justified, if only because the gross appearance of the aged disc argues against it. Furthermore, collagen[14] experiments in the rat indicate an extremely slow turnover, since the half-life of collagen is 500 days as compared to the animal's life-span of 700 days.[8]

Reproduction of Aging in Vitro

The denaturation described in relation to the intervertebral disc, is not unique in the organic world. The fate of collagen depicted in the life cycle of the disc can be matched exactly by the experimental application of heat to artificial systems. Thus a gel can be transformed into a sol state in which the protein fibers, precipitating out of solution, show increase in size and become oriented in a single plane, while the whole system shrinks in volume. Such occurrences, of course, do not mean that heat is responsible for disc degeneration in the living organism; they do signify that the final denaturation process is similar regardless of the exciting cause or causes and regardless

of the site of action, whether *in vivo* or *in vitro*. Interestingly enough an enormous acceleration of the denaturation rate of *isolated* kangaroo tendon develops with a mere rise of five degrees between 38 and 43 C.[8] However, this experiment, though provocative, is hardly to be interpreted as interdicting the application of heat to the spinal column. Further work in the intact animal would be of interest.

More convincing as an external agent because more natural and more continuously operative during life may be mechanical force in producing denaturation of macromolecules. It is known that shearing and stress forces can cause alteration in the colloidal state of proteins in the same sense as that observed with heat. The effect is necessarily slow and would be consistent with the fact that denaturation first appears in man after 20 to 30 years. Furthermore, the vertebral spine can be horizontally oriented for the play of these forces upon the mammalian disc, as exemplified in the exertions of a dog chasing his tail.

But, whether or not pertinent to the subject, pressure loading apparently is not crucial as judged from the fact that connective tissue elements of the aging dermis show the same denaturation changes. What other factors may be implicated at least for the human spine? X-rays have experimental confirmation but can scarcely account for clinical events. Certain chemicals in sizable concentration produce denaturation; but the long-range effect of drugs in living organisms has not been studied. In the present stage of our knowledge no definite answer can be proposed to account for the known changes.

Clinical Syndromes of Discogenic Disease

Whether the agents just discussed are significant only *in vitro*, or are really operative in the life cycle of the disc, connective tissue does undergo denaturation of its collagen and mucoproteins. Predicated upon this natural history of gradual biochemical and physicochemical alterations leading to deterioration of function, a rational explanation may be attempted to unify the diverse and apparently different manifestations associated with low back or neck syndromes encountered in clinical practice. This proposal may be inherently dangerous, as all too often the temptation to reduce complex phenomena to a simple denominator has led to error. On the other hand, if a concept can justly unify apparently unrelated events, avenues are opened for understanding and therapeutic exploitation. At the present time, the concepts of low back derangements recall the Tower of Babel. Aside from herniations of the nucleus pulposus, various explanations have been presented for the genesis of pain based upon (1) "sprained" muscles forming an extremely complex arrangement at various horizontal and vertical levels of the back; (2) "subluxation" or "strain" of the sacroiliac or b, lumbosacral joints; (3) "strained" ligaments of the back; (4) fascial affections vaguely termed "fibrositis", "fasciitis", "trigger points", "fibrositic nodules"; (5) "herniated fat pads" of the subfascial adipose layer. These have enjoyed or are enjoying an unwarranted popularity but they lack firm documentation. While the first three mechanisms, excluding sacroiliac affections, may account for a few cases (spondylolisthesis, although congenital rather than acquired, is definitely an example of 2, b), each leaves something to be desired as a major mechanism for low back pain.

Herniation of the nucleus alone stands unquestioned. Nevertheless — and I wish to stress this — the thesis developed here, predicated upon the natural history of the disc, suggests that back pain is often due not to herniated nucleus pulposus with accompanying radicular irritation, but rather to "discogenic disease" giving rise, aside from herniation, to diverse disturbances seated in the several structures intimately related to the intervertebral disc and all having the common factor of pain.

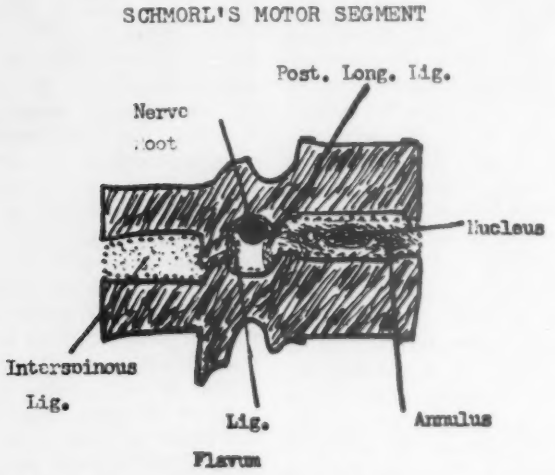

Fig. 2 — Diagram of two adjacent vertebrae with intervening "mobile segment." The apophyseal articulation is shown between the interspinous space and intervertebral foramen.

To develop further the interrelationship of aging disc, neighboring structures, and back pain — cervical as well as lumbar — attention is called to Schmorl's concept of the "motor segment".[7] In this discussion it will be termed the "mobile segment", in order to avoid confusion with specific neuromuscular meaning. Figure 2 pictures two adjacent vertebrae and the intervening mobile segment comprised of disc, intervertebral foramen, nerve root and neighboring posterior longitudinal ligament and ligamentum flavum, facet articulations, and, finally, interspinous space and ligaments. The segment should be visualized in three dimensions. Flexibility of the spine would be impossible were it not for the mobility allowed by the structures in question. On the other hand, disturbances in the dynamics of the mobile segment, consequent to narrowing or derangement of the disc, may lead to several pathologic changes, each capable of producing pain: facet arthrosis based upon unphysiologic "axis of motion" of one body upon the next,[1] ligamentous edema, nerve root irritation, or combinations of them. The disc itself is never a direct source of pain, as it lacks a nerve supply. The following section is an attempt at synthesis of the various manifestations of discogenic disease by taking into account alterations not only of disc but of physiologically and anatomically related structures.

Mechanical Displacement of the Disc

Of all syndromes related to derangement of units of the spinal column, the one most publicized is posterior herniation of the nucleus pulposus into the spinal canal with consequent compression of the adjacent spinal nerve. In the cervical region a compression myelopathy may result. In two studies of 282 extruded or protruded discs, evidence of aging was invariably found.[9,10] Since 40 "normal" (undisturbed) discs showed increasing evidence of degeneration with age, it is reasonable to presume that alterations precede rather than follow derangements.[9] The following chain of events may, therefore, be postulated. The shrunken annulus has gradually lost its resiliency, as already described, and presents breaks in its continuity. Because of impaired water-binding capacity, the swings in volume of the nucleus with alternate loading and relief, as suggested by studies of imbibition pressure, further attrition of the

annulus. Moreover, no longer elastic with decrease in hydration, the nucleus, particularly in flexion of the spine, imposes an additional strain upon the enveloping structure usually in a unidirectional, posterior thrust. The result may eventually be acute extrusion, often but not invariably with injury to adjacent nerve root, the classical herniated nucleus pulposus. Dural adhesions may form later. This condition, however, accounts for only a small number of sufferers from low back pain: in 1000 cases observed in a multidisciplinary low back clinic, only three per cent satisfied clinical criteria for herniated nucleus pulposus, subsequently confirmed at operation.[11]

The low incidence of such extrusions teaches us that, in spite of functional maladaptation and potential derangement, counter-processes come into play offsetting the tendency to frank disruption. In a large number of autopsy studies Schmorl states he has never seen true healing but the eventual sclerosis makes for stabilization of the structures involved.[7] This development will be further examined in the chain reaction of biologic aging.

Herniation of the nucleus pulposus may also be profitably considered from the point of view of incidence of site. In man, the incidence is twice as great in the cervical as in the lumbar region.[12] In dogs the entire lumbar region is involved with a high degree of frequency; the cervical portion, considerably less often.[2] These observations highlight the opinion that high degrees of flexibility may contribute to mechanical displacements. In man 80 to 90 per cent of lumbar posterior nuclear protrusions take place in the discs beneath the fourth and fifth vertebrae.[7,12] Of cervical herniations 75 per cent occur in the fourth to fifth, fifth to sixth and sixth to seventh cervical interspaces.[12] Here again the common denominator appears to be great flexibility or, seen from the physiologic view, continual stress loading upon the disc. These statistics support the notion, previously suggested, that an effective stimulus to denaturation of the structural macromolecules in the living organism may be mechanical forces: derangement always is associated with and appears to follow denaturation, and when it occurs is found in the vast majority of cases at sites where forces act excessively. To complete the argument, evidence ought to be adduced that the incidence and severity of denaturation changes in undisturbed discs are located predominantly in the same lumbar and cervical sites. If this is true, aging of normal tissue in the biologic sense results largely from environmental forces or agents acting upon a dynamic equilibrium, and not exclusively from inherent disposition. These positions are yet debatable, but other fields of study support the former one, for example, the genesis of atherosclerosis. It also is likely that inborn errors or defects will tend to accelerate the aging process; in relation to the disc, it would be of moment to correlate anatomic anomalies of the spine with presence and intensity of denaturation.

By far the commonest type of extrusion is vertical herniation of the nucleus, known as Schmorl's whorls when delineated by x-ray. Material is forced through the chondral plate into the soft cancellous bone.[1,7] Schmorl does not believe they produce symptoms. The underlying weakness allowing such protrusions is probably rooted in embryologic vestiges. The primitive notochord condenses into islands, subsequently giving rise to the nucleus pulposus; persistent strands may provide a breach in the annulus and chondral plates.[1] The important possibility remains that, although such protrusions are symptomless, the consequent narrowing of the intervertebral space, shift in axis of motion, undue stress upon facets, ligaments and annulus fibrosus, already the seat of faults, may later lead to expression of symptoms.

A third mechanical derangement has been described from postmortem material: anterior prolapse of the nucleus through the cartilaginous plate dis-

secting off a wedge of bone at the anterior margin of a vertebral body. It is considered to be asymptomatic.[7]

Finally, partial protrusion of the annulus fibrosus has been described in man and animals. With aging there occurs flattening of the annulus impaired in elasticity and resiliency. Continual battering against the posterior longitudinal ligament leads to its eventual weakening. Actually, fibers of the ligament are intimately joined with fibers of the annulus, so that the former shares in the mechanical faults of the latter. Dural compression is the result of protrusion. In the dog myelopathy has been reported, inasmuch as the spinal cord is lower in extent than in man.[2] Human postmortem material indicates that such occurrences are less than rare: 30 cases of 99 unselected autopsies revealed definite annular protrusions in ridged or nodular form encroaching upon foramen or canal, and usually "calcified".[12] The author has seen one patient whose pain may be explained by an annular protrusion. A woman aged 40 while lifting furniture developed sudden low back pain. In 48 hours a typical radicular syndrome developed in the right lower limb: absent ankle jerk, hypesthesia in the lateral aspect of the foot, pain radiating to the calf, a positive Lasègue sign, and aggravation of pain with appearance of a burning sensation around the base of the fifth toe upon coughing and straining. A body cast was prescribed for six months; but upon its removal, radicular symptoms recurred. Consequently, laminectomy was performed. No nuclear material was found; the only abnormality described by the neurosurgeon was a large, firmly anchored "exostosis" protruding posteriorly at the level of the interspace between the fifth lumbar and first sacral segments. The observation suggests a protrusion of the annulus which along with overlying posterior longitudinal ligament, eventually became ossified. The possibility is strong that such protrusions without extrusions of the nucleus can cause back pain.

Ligamentous and Articular Changes

While herniation of the disc is accompanied by changes in the other structures and spaces of the mobile segment, it is important to realize that the mobile segment is also affected even when aging occurs in a disc remaining *in situ*. Summarizing his observations on extensive pathologic material, Schmorl concludes that dehydration, desiccation, rigidity, fissuring and shrinkage in the volume of the disc produce a general "loosening of the motor segment", finally ending in various transformations of fibrosis, chondrosis, ossification, or calcification.[7] At some stage in this process, swelling of the posterior longitudinal or other ligament may give rise to "sciatic pain", since ligaments are rich in nerves. Indeed, under acute conditions such as thrusting probes or injecting formalin or hypertonic saline into interspinous ligaments, "sciatic pain" has been reproduced.[13,14] Genesis of pain could also be due to pressure upon a nerve root by edematous structures which extend laterally to overlie portions of the intervertebral foramen. Such situations would be temporary, and would explain the clearing of a radicular syndrome difficult to reconcile with a displaced nuclear fragment. A loosened mobile segment giving rise to a great degree of instability may of itself prolong root irritation with chronic radicular pain.[7] These mechanisms are postulates based on anatomic and pathologic considerations; they await clinical confirmation. Nevertheless, they are consistent with known processes and related changes in structures comprising the vertebral unit.

Another source of pain arising in the vertebral column unexplained by herniated or prolapsed disc resides in the apophyseal joint. The "facet syndrome" has long been known. Here again consideration of established facts suggests the hypothesis that arthrosis of the intervertebral joints, one of the several end-results of the aging disc, may be implicated as a source of pain. Steindler presents several possibilities.[15] Degen-

eration (aging) and thinning of the disc leads to subluxation of the apophyseal joint with degenerative arthritis and back pain. Local joint trauma could certainly eventuate because of posterior displacement of the axis of motion consequent to narrowed interspace.[1] Secondly, the "facet-synovial impingement" described by this author may develop. Here edema of the capsule, subsequent calcification and adhesion to the meningeal cover of the nerve root and osteoarthritic spurs are sufficient to maintain chronic low back or cervical pain. Steindler believes that the click on twisting is due to the underlying apophyseal subluxation. Finally, arthrosis itself may simulate radiculitis of the herniated nucleus or "sciatic pain" when osteophytes impinge on nerve roots. This apophyseal spurring is commonly found in joints lying between the third lumbar and first sacral segments. With the incidence of apophyseal arthrosis (complicated and uncomplicated) as high as 70 per cent, the possibility of impingement upon nerve root by spurs is not inconsiderable.[1,5]

In figure 3 the primary causes and primary and secondary results are summarized in three main categories: (1) herniation or prolapse of elements of the disc; (2) involvement of the apophyseal joints; and (3) changes in ligaments of the spinal column. Nine different clinical conditions are presented, all of which have been described in the literature. Six[2,4,6-9] definitely are or may probably be responsible for pain in man. It is to be noted that "radiculitis" is not synonymous with herniation of the nucleus; nor by the same token could every radiculitis entail a positive myelogram. Furthermore, in the life cycle of an individual two or more syndromes could be present, as described in the following.

A male, aged 51, developed a sudden disabling low back pain following unaccustomed physical activity. There was a history of intermittent back pain of 20 years' duration. The pain radiated to the anterolateral aspect of the lower right thigh. Numbness and paresthesias were described in the same region along with reduced perception of pin-prick and touch. The knee jerk was diminished. Symptoms cleared after 12 days of pelvic traction in bed and he was able to resume full activity. Six months later following a fall, severe, deep-seated, boring pain developed. It was localized at the level of the fourth lumbar vertebra to the *left* of the midline. No changes in reflexes or sensation were noted at this time; only limitation of motion of the lumbar spine and spot tenderness. After 32 days of traction and bed rest the pain diminished. He

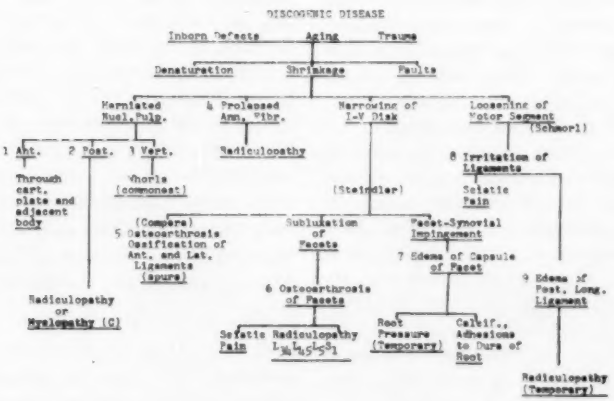

Fig. 3 — The pyramid of causes, primary effects and secondary effects.

has been on full activity without symptoms for 18 months. It would appear that the first attack was radicular in nature and cleared spontaneously, while the second attack was quite different, possibly an apophyseal arthritis. Two distinct syndromes in the same individual can be predicated upon the common ground of discogenic disease, which embraces various structures and mechanisms. Discogenic disease, therefore, refers to a background state underlying several possibilities; it is distinct from a "slipped" or herniated disc which is only one of them.

The concept of discogenic disease may also be extended to the commonly encountered syndromes of vertebral pain with ill-defined localization in a limb conflicting with dermatomal distribution. Inman has cogently stated and documented the case for "sclerotomal" patterns of pain arising in fascia, ligaments, periosteum and other mesenchymal tissue with far-reaching areas of radiation from proximal girdle to distal extremity. The pain is vague and objective neurologic examination negative.[14] This kind of "sciatica" may well arise from involvement of spinal ligaments or similar soft structures: certainly both clinical experiments and pathologic examinations support this view.[7,13,14]

There are two apparent difficulties toward accepting the view that discogenic syndromes, at various stages of efflorescence, may account for a large variety and number of cervical, thoracic and lumbar back pains. One is that symptoms occur often without x-ray changes; the other, conversely, x-ray changes occur without symptoms.

The first point invests an extraordinary degree of confidence in a technic which, though highly useful, is beset with difficulties inherent in gross vision, reduction of a three-dimensional system into a two-dimensional one, inferences from superimposed shadows, and lack of sensitivity to all but the highly opaque. Indeed, one often hears references to low back pain as if x-ray changes were the *sine qua non* of a positive diagnosis.

The truth is that pain may arise from radiolucent structures before roentgenologic changes supervene such as narrowing of the intervertebral space or ossification and calcification. In fact, without such a device as myelography herniation of a nucleus pulposus remains beyond demonstration by ordinary x-ray films. By the same token, they may not be helpful in other discogenic syndromes.

Turning to the second objection, must gross x-ray changes necessarily be accompanied by clinical symptoms and signs? If the argument holds that undetectable changes in the mobile segment may be accompanied by pain, then visible ones should increase the probability by so much the more. Yet it is well-known that striking evidence of spurring and other deviations from normal appear in routine x-ray films of individuals without past or present difficulty. This is a valid objection; but not necessarily inimical to the concept presented here. In the first place, it is inferred that similarity of the endpoint manifest by x-ray spells identity of process. It would be hardly conceivable that two vertebral columns are constructed and react exactly alike. Hence, regardless of final appearance, the clinical course may be different. Secondly, whether we can identify it or not, it is reasonable to assume that compensatory mechanisms are operative more or less effectively in individuals; otherwise back pain would be universal, as the process of denaturation of the disc is universal. In support of the tendency to adaptation, there is plentiful evidence of dynamic and structural stabilization of abnormal mobile segments in the final stage: large swings in imbibition pressure finally cease with sclerosis of the nucleus pulposus[6]; and, concomitantly, ossification of ligaments and calcification of the annulus fibrosus contribute to the same end.[7]

The possibility exists, nevertheless, that alterations in structure and composition of the disc do not precede but follow the "loosening of the mobile segment". Assuming primacy of me-

chanical derangement, it may so impair nutrition and consequently metabolism of the nucleus and annulus that eventual degeneration is inevitable. Deucher and Love found gross changes in all ages in almost every instance of 100 "protrusions" of the disc.[10] Eckert and Decker describe similar changes in 182 mechanical derangements irrespective of age, even in the second decade.[9] The force of the argument is lessened, however, when the latter authors report that in 40 discs remaining undisturbed *in situ* degenerative changes were absent in the first three decades but thereafter appear with increasing incidence. Schmorl and Junghanns present similar evidence.[7] Finally, studies on dogs and other mammals corroborate these findings.[2]

On these grounds it is difficult to escape the conclusion that "denaturation" is the primal event. The aging process appears in the young adult, progressively modifies the structural integrity and functional adequacy of the mobile segment, and thereby sets the stage for mechanical derangements of the disc, and subsequent reaction of the intimately related structures.

It is not possible, although it would be gratifying, to conclude with positive therapeutic recommendations. But, at least, an evaluation of current treatment can be considered within the framework of the concept presented. Persistence of pain following laminectomy for herniated nucleus pulposus may be due to unsuspected multiple derangements, for two or more discs may display advanced aging with mechanical disruption.[7] But this cause is infrequent. It is probable that the chain reaction set off by instability of the mobile segment, as set out in figure 3, accounts for the major portion of instances of unrelenting postoperative pain. Probably, some cases have been erroneously dubbed "psychogenic", even granting its role in chronic disabling back pain. But as long as herniated or "slipped disc" will be regarded as the sole aberration related to structures of the intervertebral space, just so long will "failures" following laminectomy be ignored, ascribed erroneously to psychologic causes, or treated inadequately. Removal of extruded disc material will not guarantee adverse reactivity of other related structures with prolongation of pain.

Recent trends may go far toward remedying surgical failures where multiple herniations are out of the question. The tendency in the past five years is to remove the whole disc in the case of extrusion of the nucleus pulposus. This procedure is followed by replacement with a bone block or removal of the cartilaginous plates to fuse contiguous bodies. Such a result should prevent instability of the mobile segment and minimize the possibility of persistent pain. Some surgeons do not even attempt fusion, if no defect exists; spontaneous fibrous fusion appears to supervene. This recourse entails a minimum in time of hospitalization and convalescence.[16]

There are non-operative procedures which appear to harmonize with the views presented here. Pain arising in connective tissue structures (e.g., ligaments) has already been discussed in reference to sclerotomal localization. These areas are tender to pressure and call to mind trigger points.[14] The practice of anesthetizing them would appear to have a firm foundation in the interruption of sclerotomal reflex pain as is the case with dermatomal patterns. Incidentally, "fibrositis"* may also be explained by the sclerotomal mechanism. It would be profitable to study carefully what Inman described over 15 years ago.[14] His contribution rests on the differentiation of pain originating in a nerve root as opposed to fascia and ligaments. All referred pain syndromes are not radicular. This distinction involves important therapeutic differences.

Nor is every radiculitis, moreover, caused by herniation of the nucleus pulposus, or osteophytic spurs situated near the foramen. Impingement by

*This term is meant to exclude the fibrositic nodules or other manifestations of rheumatoid arthritis.

edematous ligament or synovium should be considered, even in the face of fibrillation potentials in the corresponding myotome. In my experience, this conclusion is difficult to disregard when later the clinical electromyographic abnormalities have vanished. As already mentioned, temporary radiculitis has been predicated on pathologic grounds.[7] If this mechanism is acceptable, therapeutic implications would favor initial non-operative management, unless muscle atrophy and sensory changes are so striking that the suspicion of massive nerve compression is inescapable. In case of doubt, myelography properly done would resolve the dilemma. It is difficult to ascribe a mechanism other than ligamentous or synovial impingement upon a nerve root when examination of a patient presents positive Naffziger and Lasègue maneuvers, absent ankle jerk, decreased sensibility in the outer aspect of the foot, accompanied by a completely normal myelogram. This type of patient subsequently returns to work. Perhaps, many needless laminectomies with their toll of disability would be avoided if radiculopathy associated with low back pain were not automatically regarded as herniated nucleus pulposus.

Another non-surgical procedure is exercise. In the experience of many its use is beneficial in the individual with no frank evidence of neurologic deficit. Possibly, increased muscular strength and consequent improved axial balance reduce instability of the mobile segment. In a structure the elements of which are highly mobile, the supporting and mediating musculature must play a role in the over-all functional integration of related units. Attention is particularly called to the multifidi muscles connecting adjacent vertebrae.

In the realm of pure speculation is the therapeutic connotation of hormones for low back disorders. Experimentally, endocrine changes have been linked to depolymerization of polysaccharides; while hydrocortone inhibits their metabolic turnover.[2] Whether connective tissue alteration in general is also a function of the endocrine life cycle remains for future decision.

Finally, more as a matter of theoretic interest than clinical applicability is the experimental acceleration of denaturation of connective tissue ground substance and collagen by heat. This interesting relationship *in vitro* may perhaps be elucidated in chronic experiments upon the intact animal.

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We here in America, hold in our hands the hope of the world, the fate of the coming years; and shame and disgrace will be ours if in our eyes the light of high resolve is dimmed, if we trail in the dust the golden hopes of men.

– THEODORE ROOSEVELT

Comparative Outcomes of Respiratory Poliomyelitis Patients Treated from Onset and in the Chronic Phase of the Disease

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● From a total case load of over 2,500 poliomyelitis patients the authors observed and treated 199 acute and 246 chronic poliomyelitis respiratory patients through 1959. These 445 surviving respiratory patients have been consecutively admitted and all have been treated under the same program so that some comparisons may be made. In this report the authors have examined the comparative outcomes from the points of view of: (1) respiratory aid emancipation; (2) residual muscle strength estimates and capability for independent function (feeding, hygiene, communication, etc.; (3) range of motion; and (4) some aspects of sequelae and medical complications. The results seem to indicate a more favorable outcome for the patient who has comprehensive management available from onset. This is evidenced by: (1) 84 per cent of patients managed from onset are safely freed of their breathing aid at first discharge contrasted with 20 per cent of those transferred to the Center in the chronic phase of the disease. (2) Muscle test strength estimates show a striking difference in the two groups. The group managed from onset shows an average strength increase of 22 per cent in the first year after onset and over one-half the patients have 70 percent or more residual muscle strength (recovery) at one year or later from onset. Less than one-fourth of the chronic patients showed greater than 50 per cent residual total body muscle strength (recovery) over the same time span. (3) There is a significant difference in residual flexibility (range of motion). Twice as many of the chronic patients had tightness about joints that limited function as did those managed from onset. (4) Likelihood of survival evidenced by long-term mortality of the patients seen late was over three times that of patients managed from onset. Discussion of some of the factors likely to account for these differences is presented.

The availability of Salk Vaccine protection against paralytic poliomyelitis apparently has resulted in a decreased incidence of the disease. In 1959 there were over 90,000,000 citizens of this country who had not availed themselves of this protection. Of these, more than 40,000,000 were under the age of 40 years and nearly 21,000,000 under the age of five years. In this latter group, 30 per cent were not vaccinated, and an additional 22 per cent have received fewer than the recommended basic immunization of three doses. This constitutes a vast pool of susceptible persons and this Center's 1958-1959 experience would indicate that the "polio problem" is far from being solved. On a national basis there were 5,694 paralytic cases reported for the year 1959, an increase of 82 per cent over 1958.[1] There are over 300,000 individuals alive in the nation who have had paralytic poliomyelitis. Within this number there were at least 50,000 classified as having severe paralysis and 1,756 still using breathing aid in 1957. The number who used breathing aid at the onset of their disease is not known.[2] The numbers have been added to in the past three years.

Since 1950 the National Foundation, in cooperation with various medical schools and medical centers, has established 16 regional respiratory and rehabilitation centers. These centers have

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gathered together a corps of highly trained personnel from the various medical and paramedical specialties who are skilled in the management of the respirator or otherwise severely paralyzed poliomyelitis patients. All have been developed in association with academic medical programs and have teaching and research activities in addition to patient care responsibilities. To date, more than 4,200 of the most severely involved of these patients have been seen in the regional respiratory and rehabilitation centers. This report will examine some aspects of the first 10 years' experience in the care of respiratory poliomyelitis by one of the older and larger regional respirator centers.

There have been many publications emanating from the centers about specifics of patient care but rarely has there been a published attempt to answer the question of whether a comprehensive care program of this type was worthwhile. Although this may be self-evident to those concerned in such programs a systematic study is still needed. This type of examination is made in this report because this Center has been unique in accepting large numbers of both the acute and chronic poliomyelitis respirator patients. A comparison of individuals who ultimately end up under the same program should shed some light upon the medical, social, and economic values of a respirator center's comprehensive care program. These people have been evaluated from the point of view of their comparative respiratory outcome, residual muscle function and strength estimates, range of motion, and certain aspects of mortality. These are but a few of the parameters studied but are the ones most readily reviewed and submitted to descriptive analysis. Although the chosen criteria are helpful the "outcome" should be complemented by a composite judgment of the patient's, the family's, and the personal physician's evaluation of whether or not there was anything accomplished by hospitalization and continuing care and follow-up.

The Southwestern Poliomyelitis Respiratory and Rehabilitation Center* has followed the patients treated from onset for an average of 60 months and those of the chronic group have been followed for an average of 65 months. It is our belief that this is a sufficient length of time for achievement of near maximum advantage from therapeutic procedures to be evaluated.

Definitions and Description of Experience

The word "acute" in this study refers to patients who were treated at the Southwestern Poliomyelitis Respiratory and Rehabilitation Center from the onset of poliomyelitis. An acute respirator patient is defined as one who required breathing aid for a period greater than two weeks. Where the term "chronic" is used, it refers to a patient who was treated during his acute and immediate post-acute phase in a hospital other than the regional center. Patients admitted in the chronic phases of the disease had spent an average of six months hospitalization elsewhere before referral to the Center. The outcome of 445 consecutively admitted, surviving respirator patients is included. Of this number of patients, 199 were treated in the Center from the onset of their disease (acute) and 246 treated during the later phases of illness (chronic).

The 199 respirator patients requiring respiratory assistance for longer than two weeks from the onset of poliomyelitis are the number of individuals with chronically impaired breathing accruing from 979 acute paralytic poliomyelitis admissions. During this period there were 1,236 patients diagnosed as having acute paralytic or non-paralytic poliomyelitis.

Figure 1 has been prepared in order that similarities and differences of age distribution of patients followed from onset and in chronic stages of polio can be identified. The age group distribution of acute and chronic respirator pa-

*Now incorporated in the Texas Institute for Rehabilitation and Research.

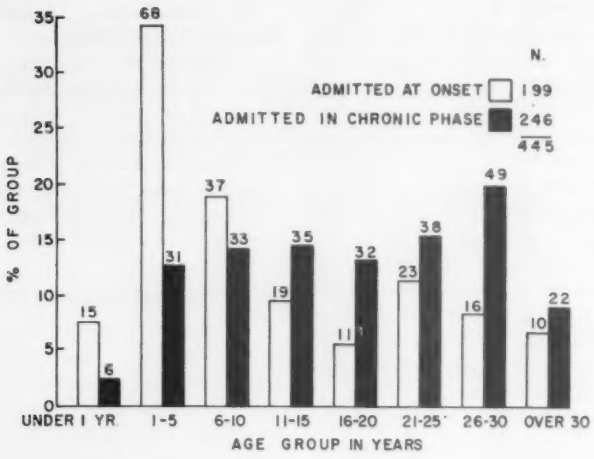

Fig. 1 — Age group distribution of poliomyelitis respiratory patients. This figure shows the age distribution of patients related to age at onset of poliomyelitis. The clear bars represent patients treated in the Center from the onset of disease and the shaded bars represent patients admitted in the chronic phases of poliomyelitis. The horizontal axis indicates the various age groups and the vertical axis shows the percentage occupied of the acute and chronic groups respectively by each age group. The number at the top of each bar is the number of patients in that group. (See text for definition of acute and chronic.)

tients is represented with the vertical height of a bar graph indicating the percentage of the total population for a particular age group. The Center has treated more acute patients in the younger age groups and the chronic patients were most often from the older age groups. Yet the spread of age range and the number in each group is not so different but that some comparisons of the acute and chronic groups can be made. Sex and race are not indicated because they do not appear to be related to differences in an individual's response to his disease within the chosen points of comparison. This is not true of the impact of chronic disease of this severity upon the family where age and sex of the person appear to be significant.

General Characteristics of the Treatment Program

The treatment program for a patient is completely individualized, but over the years a pattern has developed.[3, 4, 5] On admission of a chronic patient, or at the onset of illness of an acute patient, the individual is evaluated from a number of points of view. These include his general physical condition, nutritional state, strength estimation, degree of functional independence, orthotic and appliance needs, and, as indicated, a review by the orthopedic surgeon, physiatrist, urologist or other medical specialist of his particular needs. Medical social service contacts with patient and family are constant. These evaluations are repeated no less often than monthly and the medical and paramedical professions directly concerned with a patient's treatment meet together at weekly intervals under the direction of the responsible attending physician. At these weekly conferences the patient is discussed by each professional staff member from his particular point of view, and at the close of discussion a joint decision is reached regarding any indicated program changes and who is to do it. This constant monitoring of the treatment program permits close integration of all the services needed by a severely involved patient and his changing tolerance to physical treatment; it also

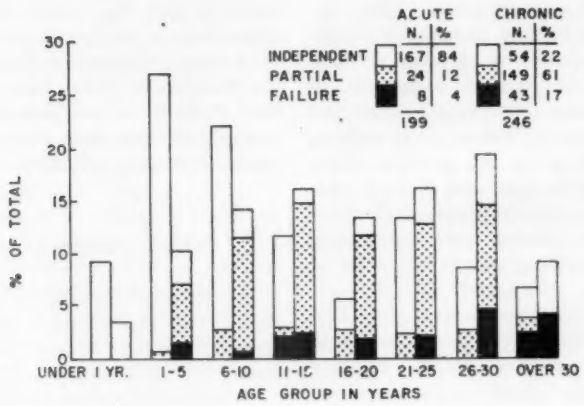

Fig. 2 – Comparative respiratory outcome of 199 acute and 246 chronic respiratory poliomyelitis patients. The horizontal axis reflects the age groups and the vertical axis represents, in percentages, the patients within each age group who were successfully freed of all breathing aid, partially freed of breathing aid, and those who continued to require full-time breathing aid. (See text for definitions of independent, partially independent and failure.)

makes possible early recognition of problems, both medical and psychosocial.

In pacing a program it is customary to introduce but one change at a time. For example, decrease of breathing aid, increase in sitting time and functional activities, and beginning ambulation are carried out sequentially rather than together. Each change of any magnitude is followed by one or two weeks of close observation to be certain that the demand on physiologic reserves has not been excessive. The speed of emancipation from breathing aid is often sacrificed to permit neuromuscular physical rehabilitation to proceed without recurrent interruption.

For purposes of description it is easier to outline outcome results by specific categories. This has been undertaken with a full understanding that for an individual patient it is a composite of these and other evaluations which determines his over-all situation.

Respiratory Outcome

The degree of freedom from artificial breathing aid for the acute and chronic patients was compared using the date of initial discharge from the Center as a convenient point of time. The following definitions were used to define the status of each patient.

Independent–Does not require any type of breathing aid.

Partially Independent–Requires breathing aid, but less than 22 hours a day and generally does not require the use of the tank respirator.

Failure–Requires aid 22 hours or more a day. In general, use of the respirator during the day is not required, but it may be used at night. Patients are placed in this group who utilize glossopharyngeal breathing to gain otherwise unattainable unaided breathing time.

Freedom from breathing aid is not necessarily a primary goal in the Center. Respiratory aid is used judiciously to permit the patient the maximum amount of independent or natural breathing his respiratory endurance allows, taking into account his over-all activities. For example, we believe it is much better for a patient to sleep in a breathing aid and to be able to pursue a vocation during the day than to expend all his energy to achieve total independence from breathing aid and then be unable to carry on any other activity such as home-making, educational efforts or vocation.

Figure 2 was prepared, using the above definitions, to investigate the respiratory outcome in degrees of freedom from respiratory assistance in each age group, contrasting acute and chronic patients. There is a striking difference in these two groups of patients. Eighty-four per cent of the people who were treated comprehensively from the onset of disease were completely freed of breathing aid at the time of discharge as contrasted with 20 per cent of the patients treated late. The weaning failure group is of considerable importance. All the weaning failures in the acutely treated patients occurred in two age groups, namely, the adolescent and the adult. The subjective impression of personnel assisting these patients is that the adolescent and the young adult presented the most difficult clinical problems. For example, there was a greater incidence of metabolic and renal problems in these two age groups. Weaning failures occurred in all of the age groups of chronic patients.

One logical objection to a comparison of a group of patients who have their disease treated from onset in the same place with a group treated in different places is that the chronic patients referred to the Center are but the residue of the extremely involved group. If the case load figures reported to The National Foundation for the operational period of this Center are considered and the assumption is made that an equal number of favorable outcomes were present among individuals who were not admitted to the Center, then there should have been four times more patients freed of respiratory aid than were reported, and five times the number of partially independent patients who had used respirators. Although adequate statistics are not available to test this hypothesis, the bulk of respirator patients in the southwestern region of the nation found their way to centers or did not survive the "threat-to-life" phase of poliomyelitis. Doubtless, too, some people were not put in respirators who might have been if they had been treated from onset in a center. Yet in our experience with acute patients less than 10 per cent of the people going into respirators had a form of self-limited breathing irregularity that does not require continued breathing assistance.

Muscle Strength Recovery

It is helpful to evaluate the outcome of the two groups from the point of view of tested muscle strength recovery.* Four hundred and ninety consecutively admitted acute paralytic patients were tested for muscle strength. This number included 133 respirator patients. Comparison of the respirator and non-respirator patients shows that the extent of involvement of the respirator group extends all the way from practically no residual involvement to almost total paralysis. The slope of the curve for the over-all experience fits the pattern of severity of involvement that has been nationally experienced and which would be expected from the protean character of the disease.

If a group of respirator patients is compared in tested muscle strength as soon after onset as possible and again one year or more from onset, it is possible again to identify the initial pattern of weakness in contrast to the pattern of recovery of muscle test strength. The mean percentage recovery in tested strength for the respirator

*Numerical values for the manual muscle test were arrived at by the following procedure. An arbitrary value of 1000 points was chosen to represent test normal strength of the muscles usually tested and graded, plus the diaphragm leaves and intercostal muscles which can be graded by fluoroscopy. The muscles of the head and swallowing mechanism are eliminated. It is recognized that "test normal" does not necessarily represent "normal" power and endurance. Approximately equal values were assigned to the three primary divisions of the body (trunk, the upper extremities and the lower extremities). Then each muscle or group within the primary division is assigned a factor dependent chiefly on its bulk. (In general this factor assignment follows the expected anterior horn cell innervation ratio.) Test letter grades were assigned values ranging from five for test normal to 0 for test zero. Multiplying this value by the factor for the individual muscle gives a numerical meaning to the muscle test. The sum of these scores gives a numerical meaning to the whole muscle test. The reproducibility of this test technic and retest variability was determined and it was found to be reliable for clinical use.

group was 22 per cent. The vital importance of early protection of any and all muscle strength for its functional potential is thus established because a muscle graded "Poor" may well become a functionally useful "Fair +" if not prevented from doing so by over-zealous and injudicious physical therapy or disuse atrophy or contractures. Similarly, the effects of infections, dehydration, and the various other complications may be, in part, preventable and such complications seem to affect outcome. The respirator patient group treated from onset, in contrast to the patients who were treated later in their illness, had a very striking difference in residual muscle strength (tested one year or more after onset). Well over half the patients treated from onset have 70 per cent or more residual muscle strength, whereas practically none of the chronic patients have more than 50 per cent estimated normal body muscle strength. The degree to which selection of cases with severe involvement accounts for this difference in the chronic group is, of course, unknown. The degree to which we have included individuals that would ordinarily have been kept out of the respirator is also unknown. However, those included did require at least two weeks of respirator assistance and, therefore, the bulk of the less severely involved have in fact been excluded from our acute patient figures. Suitable criteria of comparability for statistical significance testing could not be assumed to be present and such testing was not performed. Only over-all group comparisons seem to be worthwhile and justified.

Residual Range of Motion

Another valuable criterion of the effectiveness of treatment is that of the residual flexibility (range of motion) of the extremities and trunk of the respirator or other severely paralyzed poliomyelitis patient. Figure 3 contrasts the residual flexibility of 144 consecutively admitted respirator patients in each group, and it is immediately clear that the chronic patient has higher levels of "tightness" than those treated from the onset of the disease in a center. This can be a result of over-stretching as well as under-treatment. When extreme weakness prevails around a joint, stretching to full range of motion may not be indicated. Wherever definite improvement of function or the expectation of prevention of deformity is present, it is quite proper to permit elective tightnesses to occur. A large number of the chronic patients who have severe degrees of tightness have a history of early severe painful stretching. This can cause injury to the muscles and tendons and may result in untreatable tightness. A number of chronic patients have recited the terror they felt at the approach of the physical therapist. Severe stretching is contraindicated on both physiologic and humanitarian grounds.

Functional Independence

Functional ability and independence of a patient are testable. Muscle strength estimates provide an index of the muscle strength available for function but do not give an adequate indication of what the functional ability of any patient will be in activities of living. In order to make better estimates, a functional test was devised that gives a numerical value for 50 stationary and moving activities which were chosen arbitrarily to represent functional independence. The values assigned for each activity graded were arrived at by measuring the amount of time it took someone else to perform each particular activity for a patient.

No reduction in score was made if a patient could be graded independent using assistive devices. Independence does not necessarily mean "normal" or pre-illness status, but it does mean that the individual can carry on his usual daily activities without the aid of another person. Figure 4 verifies the large differences between 121 acute patients and 132 chronic patients in functional independence. These pa-

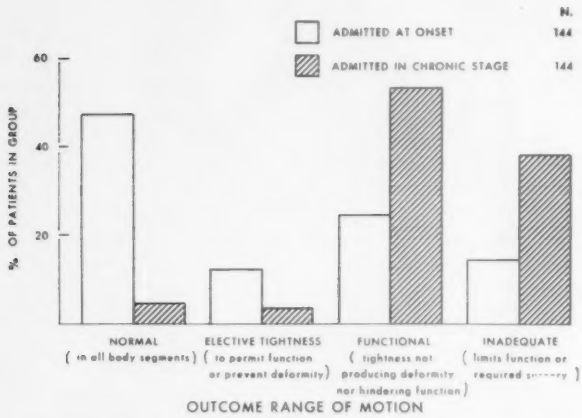

Fig. 3—Comparative residual flexibility (range of motion) in 144 respiratory patients admitted at onset and 144 admitted in chronic stages of poliomyelitis. The horizontal axis reflects the categories of tightness as determined in this Center and vertical axis shows the percentage of each group that falls into the four categories. It is apparent that the chronic patient tends to have significantly higher degrees of tightness of a level that more frequently interferes with function, or that requires surgery to be successfully released. The bar showing elective tightness indicates that more Center patients are permitted such tightness.

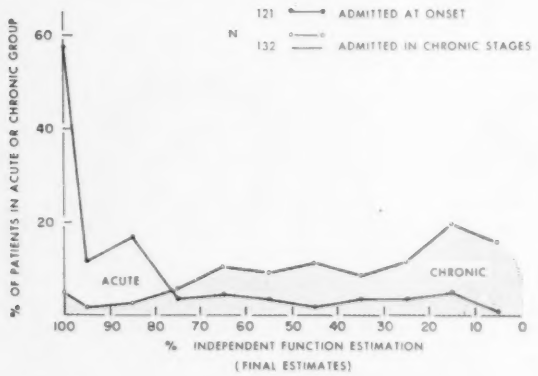

Fig. 4—Comparative outcome of functional independence for 121 respiratory patients admitted at onset and 132 respiratory patients admitted in chronic phase of poliomyelitis. The horizontal axis shows the percentage of estimates of independent function and the vertical axis shows the percentage of patients falling into each percentage group. See text for description of how the functional estimation is made.

tients were admitted consecutively. It is clear from these data that over 70 per cent of the patients admitted from the onset of poliomyelitis had 90 per cent or more independent function contrasted with less than 10 per cent of the chronic patient group achieving the same levels of independence. Small children were excluded from this analysis.

The achievement of these high levels of independent function in the group managed from onset is believed to be the summation of many factors. Included are the careful provision of every aspect of medical and nursing care during the "threat-to-life" phase, detailed medical monitoring of the pace of the treatment program and emancipation from breathing aid during con-

valescence, careful preservation of personal dignity, availability of an active orthotic program, and the continuous assistance of medical social service in maintenance of the family's interest in the patient, understanding of the purpose of hospitalization and preservation of the family solidarity.

Survival

The problem of comparing fatalities for the two groups is difficult because of the paucity of information available about the patients who died during their acute phase in other institutions. However, if one excludes from the experience of this Center deaths that occurred within one month from onset of illness, then a mortality rate of five per cent of the acute group is found during the follow-up period of 60 months. This contrasts with an 18 per cent mortality for the chronic patients. Approximately half of the deaths occurred during readmissions to the Center for intercurrent complications and the others were about equally divided between home and the community hospital. Inquiries about the deaths that occurred at homes or in other institutions were directed to the attending physician, the family, and on occasion to the hospital or the sponsoring chapter of The National Foundation. Of the deaths that occurred away from the Center a number of hospital charts were reviewed and in a few instances it was possible to get autopsy protocols.

The leading cause of deaths for both groups was respiratory infection and it accounted for 75 per cent of the deaths about which adequate information could be obtained. Renal infection and calculus formation accounted for the bulk of the remaining causes. A small miscellaneous group of fatalities resulted from automobile accidents, drowning, etc.

The personnel of the respirator centers are keenly aware of the insidious nature of respiratory insufficiency and the need for prevention of respiratory failure that can quickly overwhelm the person with residual respiratory muscle paralysis when he has a mild respiratory or urinary tract infection. This clinical dictum is not widely appreciated. For the patient who has severe weakness or paralysis of the abdominal musculature a common cold can be a fatal disease by virtue of the fact that such persons have very ineffective coughs and unless early and specific treatment of the infection is coupled with adequate artificial respiration and mechanical cough maneuvers, pneumonia and atelectasis often ensue within hours. Treatment of such situations is at best unsatisfactory while preventive measures are reasonably easily carried out and are readily available in most hospitals. In light of these findings there is obviously a need for extreme awareness of this hazard and for an educational program that should be developed primarily for the family and for the attending physician in the home community.

Further studies into other body disturbances are being pursued in this complex problem of treatment evaluation. These are currently active in the general fields of cardiopulmonary and renal disturbances and studies of the influence of psycho-social factors on outcome.

General Considerations and Summary

1. General comparative statistical experience can be used only for inference. A true alternate treatment, no treatment or placebo regimen cannot be constructed in the respirator patient care situation. It would appear that along several dimensions there is clear descriptive difference between the patient treated from onset in a respirator center and those brought in long after onset of poliomyelitis. The group treated from onset experiences more favorable outcomes in greater frequency by age and in more ways than the chronic group. There is little doubt that case selection among those admitted late accounts for some differences. This alone does not explain all

of the differences or their degree when considered from several indices of outcome.

2. A number of factors should be identified that point out the inadequacy of simple statistical treatment without proper or obtainable controls to indicate causes or reasons for observed differences. These include:

a. The deleterious effect of the summation of some preventable insults, such as deterioration in residual muscle strength, joint and connective tissue contractures, pulmonary, cardiovascular, metabolic and renal complications or the influence of psycho-social problems.

b. The patients treated comprehensively may benefit by the elimination of social regression and preservation of the motivation for acceptance of treatment and its long drawn out procedures when medical social service activity is available from onset and the strength and solidarity of the helpful family unit is thereby preserved.

c. The respirator center environment provides an opportunity for one patient to influence the other; for developing of common disease insight and for answering hidden fears and misunderstandings. These conditions may alter disease response and the patient's willful determination to follow through on medical recommendations.

d. The effect of staff teamwork in providing adaptable and diligent treatment efforts may result from attitudinal experience stimulated by the presence of many severely disabled patients. This effect is not readily measurable, but may greatly influence the treatment situation. When one faces a whole group of patients generally considered "hopeless" the whole outlook may change.

e. The influence of simultaneous research and teaching activities among a medical and allied professional staff upon improvements in the caliber of treatment must be reckoned with.

f. Changing patterns of medical management and progressive improvements alter the comparability of outcome collected together over an extensive period. Uncited and progressively improved mortality figures of the management of acute threat-to-life disease clearly fit this implication. In recent years the employment of respiratory assistance has been sustained because patients in the partially independent group often increase tolerance to functional activities if respiratory aid is continued. Thus, even in the chronic patient group a large incidence of partial independence from breathing aid may include some individuals who would otherwise be free of respiratory assistance if this were the sole objective of center treatment.

3. It is likely that there is a need for two large developments. One includes more fully descriptive comprehensive evaluation from time to time in the same individual in order to regulate treatment more precisely on the basis of sound knowledge of the natural history of the complex disease state; and secondly, the development of disability evaluation that will permit some group comparisons which will yield knowledge of cause and effect relationships in disease processes and bodily responses to the disease state. Unquestionably, respirator center care, however evaluated, has shown the importance of comprehensive medical care for the respirator patients. The sooner this type of care is available the more favorable the outcome appears to be. Although proof cannot be found in statistical methods alone, the effectiveness of respirator center programs that in some way alter comparative outcomes of the early and the late treated patient appears to be established.

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Etiology of Decubitus Ulcers: An Experimental Study

Olgierd Lindan, M.D., Ph.D.
Cleveland

● The concept of assessing the relative significance and mode of action of various factors contributing to decubitus ulcer formation is presented, using an experimental approach. A "decubitus threshold pressure" has been established for healthy animals, i.e. a minimum degree and duration of pressure resulting in well defined tissue changes leading to necrosis. A "minimal pressure lesion" in healthy man, showing minimal but specific structural and functional alterations in the skin, due to pressure alone, has to be defined. Using these two standard pressure indices the role of contributing factors (metabolic, infective, neurologic), resulting in increased sensitivity of tissue to the effect of pressure, can be examined. A method is described for measuring the external pressures to which a patient's skin is subjected in hospital circumstances, and the "pressure isobars" so obtained are illustrated. An animal model has been devised, using rabbits' ears, whereby a constant tissue response is obtained according to the duration and magnitude of pressure applied.

Decubitus ulcers continue to present a serious problem in the management and rehabilitation of the chronically ill patient despite the knowledge that, in theory, such ulcers should be entirely preventable. The degree and duration of pressure to which the patient's body surface is subjected, when in bed or in a wheelchair, is recognized as the primary factor in their causation. If this pressure is high enough to interfere with tissue circulation and if it continues for a sufficiently long time then it will result in skin breakdown. Yet one of the most baffling features of the problem is the variation in susceptibility among patients to this effect of localized pressure. Clinical observations give evidence that this susceptibility is particularly increased in the debilitated, the undernourished and anemic, the paralyzed, the infected and the feverish. If the skin area on which such a patient is resting is soiled with feces or urine, or is traumatized by sheet burn or excoriated by adhesive tape, then decubitus formation is further hastened. The precise mode of action and the relative importance of these general and local contributory factors in the production of decubitus ulcers have to be estimated in order to plan a more successful approach to their prevention and early healing.

Logically, the response of healthy tissue to external pressure per se should be assessed first without any other modifying pathology. The ranges of pressure to which the human body is subjected in health and disease must be measured and this knowledge utilized in experimental studies on tissue response to pressure. Varying degrees and duration of external pressure can be applied to the skin of laboratory animals and man and detailed observation made as to the functional, histologic and biochemical changes occurring both in the superficial and in the deeper tissues.

An attempt is being made to define in healthy animals a minimal degree and duration of pressure (a "Decubitus Threshold Pressure") which will consistently result in well defined pathologic tissue changes leading to necrosis. It is necessary also to establish in the healthy man a "Minimal Pressure Lesion" showing minimal but specific structural and functional alterations in the skin and subcutaneous tissues due to pressure alone, which will be reversible and reproducible. Using these two standard pressure indices the role of contributory factors may then be assessed. The relative importance of a particular factor will be measured by the degree to which either the standard time of exposure to pressure, or the standard level of the applied pressure, can be reduced and a predetermined lesion still produced.

The following describes the attempts so far made to pursue this line of inquiry into the etiology of decubitus ulcers.

Assistant Professor of Medicine, Western Reserve University Medical School; Director of the Metabolic Ward, Highland View Hospital for the Chronically Ill.

External Pressure in Man*

When analyzing the effectiveness of external pressure in producing tissue pathology two parameters must be considered: (1) the magnitude of the applied force per unit surface and (2) its duration.

Whether standing, sitting or reclining the body presses on the supporting surface with a force proportional to its weight. With increasing body size the increase in skin area lags behind the increase in weight and therefore the bigger the man the greater will be the pressure force per unit of skin area. The malleability of the surface on which the body rests is another major factor in modifying the magnitude of the pressure at any given point; a more pliable support will increase the area of contact and will allow a more uniform distribution of weight per unit of surface.

One easily can calculate that some of our skin areas and underlying tissues are subjected to remarkable pressure forces in the course of ordinary activities. For example, the undersurfaces of a man's feet when walking must bear pressure several times in excess of his systolic blood pressure. Yet it is possible to walk for many miles without developing any significant injury to the feet. This is due to the fact that this pressure, although high, is of short duration and intermittent.

Actual measurements, however, of the external pressures exerted over particular areas of the body have been few. Kosiak et al[1] evaluated the pressure as a factor in the production of ischial ulcers and reported measurements obtained at 12 points under the buttocks of a seated subject. The method they used consisted of flat rubber air valves, measuring two centimeters by one centimeter, located under the buttocks and connected with a reservoir of compressed air. The minimal pressure under which the air escaped from a given valve was taken as being equal to that exerted by the buttock at that point. The authors correlated the distribution of pressure with various types of chair seats. The highest pressure, over 300 mm. Hg, was recorded under the ischial tuberosities when the subjects were sitting on a wooden office chair. This high pressure was reduced to 160 mm. Hg when the seat was padded with foam rubber two inches thick.

Unfortunately this method of air valves does not lend itself easily to large surfaces and many points of measurement. In order to get a general idea of the ranges of pressure and of the body areas over which they are exerted under varying conditions, a device using the principle of spring compression has been constructed. It consists of a "bed" of perforated plywood with three inch nails supported on calibrated springs and placed in the holes 1.4 to two centimeters apart. When a body is reclining or sitting on the nail heads, the points of the nails protrude through the under surface of the board, and by measuring the length of each protruding nail the degree of spring compression can be calculated and equated with the average pressure exerted on the skin area supported by that nail (fig. 1).

Fig. 1 — Experimental "bed" using the principle of spring compression for measuring external pressures exerted on the surface of the human body.

*In collaboration with R. M. Greenway, Ph.D., and Janet M. Piazza, B.S.

In this way measurements have been made at about 1000 points on the surface of a reclining subject and about 150 points on the buttocks of a seated subject. By connecting points of identical pressure, isobars can thus be drawn. These isobars give information concerning (a) the magnitude of the pressure, (b) the position, shape and size of the area over which it is exerted, and (c) the steepness of the gradient at points of pressure variations.

Figures 2-5 and tables 1 and 2 illustrate the pressure distribution over the skin of a healthy male, age 29 years, 164 cm. high and weighing 66 kg. Two types of springs were used. The stiffer

Table 1: Range of Pressure Exerted over Various Skin Areas by a Body of a Healthy Man Lying on a Sprung Surface (For spring compressibility see the text).

Position of body and skin areas	Range of pressure mm. Hg	Pressure Area Using softer spring B cm.²	Pressure Area Using stiffer spring A cm.²
Supine			
Posterior chest wall	10 - 20	350	330
	20 - 30	255	190
	30 - 40	5	120
Buttocks	10 - 20	170	240
	20 - 40	290	165
	40 - 60	65	85
	60 - 70	0	25
Calf*	10 - 20	90	100
Heel*	10 - 30	—	7
	30 - 50	—	13
Prone (feet hanging free)			
Anterior trunk wall ...	10 - 20		690
	20 - 30		275
	30 - 40		75
	40 - 50		30
Thigh*	10 - 20		235
	20 - 30		30
Knee*	30 - 50		13
	50 - 60		5
Shin*	10 - 20		30
	20 - 30		10
On left side			
Hip	30 - 40		125
	40 - 50		50
	50 - 60		35
	60 - 70		25
	70 - 80		20
	80 - 95		10

*Mean area of right and left leg.

Table 2: Range of Pressure Exerted on Skin of Buttocks by a Healthy Man Seated on a Sprung Surface (using spring A)

Range of pressure mm. Hg	Pressure Area with feet hanging free cm.²	Pressure Area with feet supported* cm.²
10 - 20	175	220
20 - 40	265	310
40 - 60	390	175
60 - 70	65	—
60 - 80	—	85
80 - 100	0	17
100	0	4

*Feet supported on a balance pan with a counterweight of 12.5 kg.

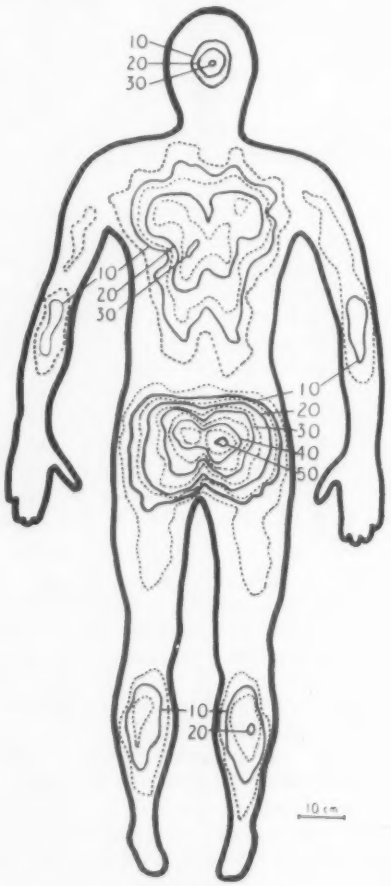


Fig. 2—Pressure distribution (mm. Hg) over the skin of a healthy adult male lying in the supine position (hands prone). Data obtained from the experimental "bed" with soft springs B. See text.

of the two (A), when placed two centimeters apart, resulted in a "discontinuous surface" compressible to a depth of one millimeter by a pressure of 4.2 g/cm.^{2} (3.1 mm. Hg). This corresponds roughly with the degree of compressibility of one and three-quarter inch foam rubber as used in this hospital for padding hard seats. The second type (B) had twice the compressibility under the same pressure.

Comparison of the isobars in Figures 2 and 3 and the pressure data given in table 1 for the supine position demonstrate that the harder surface:

(a) increased the maximal pressure,

(b) decreased the area of lower pressure contact,

(c) increased the area of higher pressure contact, and

(d) increased the steepness of the isobar gradient.

With this particular subject in the supine position (using spring "A"), the following data were obtained: on the buttocks, 40 to 70 mm. Hg over an area of 110 cm.^{2}; on the heels, 30 to 50 mm. Hg over 13 cm.^{2}; and on

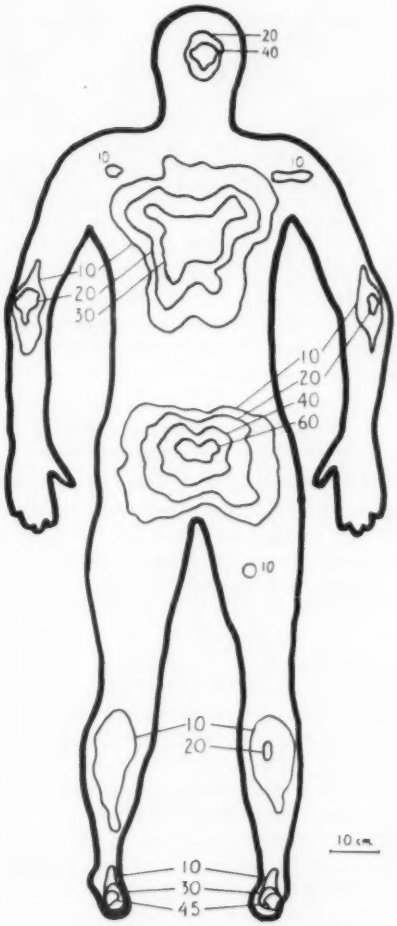


Fig. 3 — Pressure distribution over the skin of the same subject as in figure 2, also in supine position. The experimental "bed" fitted with stiffer springs A.

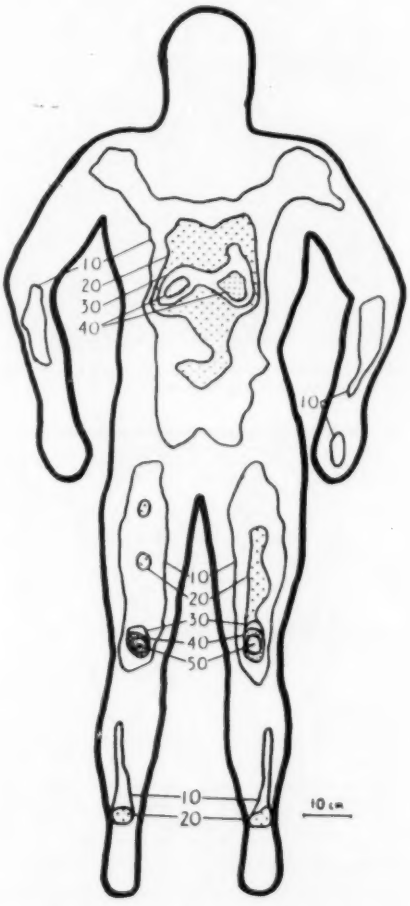


Fig. 4 — Pressure distribution over the skin of the same subject as in figures 2 and 3, in the prone position (hands supine). Springs A in use.

the posterior chest wall, 30 to 40 mm. Hg over 120 cm.^{2}.

In the prone position the highest pressure was obtained over the knees, namely 30 to 60 mm. Hg, over 18 cm.^{2}, and the next highest pressure was 30 to 50 mm. Hg over 105 cm.^{2}, overlying the costal margins.

When the subject was lying on his left side the high pressure over his hip was 70 to 95 mm. Hg, over an area of 30 cm.^{2}.

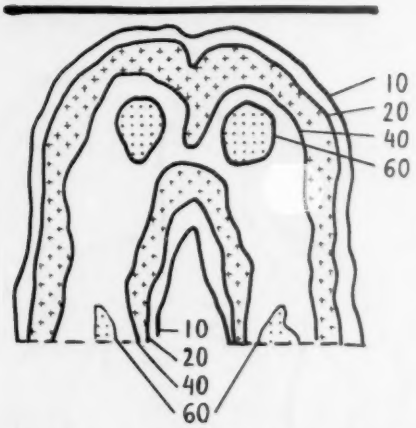


Fig. 5a—Pressure distribution (mm. Hg) over the skin of the buttocks of the same subject when sitting with the feet hanging free. Springs A in use.

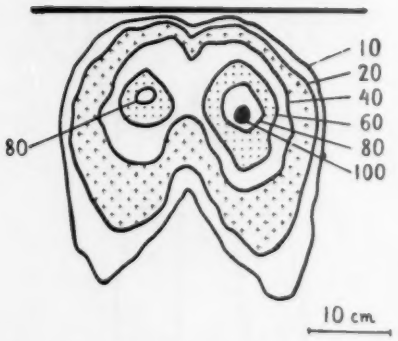


Fig. 5b — Same as figure 5a but with the feet supported on a balance pan with a counterweight of 12.5 kg.

Figs. 5a and 5b illustrate the degree to which the maximal pressure exerted over the ischial tuberosities in a seated subject is influenced by allowing the feet to hang unsupported.

Taking the mean capillary pressure as being equal to 25 mm. Hg, one can see from the given diagrams (figs. 2-5) the size and location of the vulnerable skin areas where the external pressure is in excess of 30 mm. Hg and where the pressure gradient is steepest. Thus, even with subject lying on a sprung surface which is more compressible than the ordinary hospital bed, an impairment of blood microcirculation easily may occur. This impairment, if sufficiently prolonged in a susceptible patient, could then result in the formation of decubitus ulcers. It must be borne in mind, moreover, that these pressure data were obtained on a healthy young man with good muscular tonus and adequate panniculus adiposus, and it remains to be seen how the size and distribution of higher pressure areas will differ in the debilitated subject and in patients with neuromuscular defects.

It appears that the highest pressure to which a patient's skin is subjected in ordinary hospital circumstances is in the vicinity of 100 mm. Hg. The effect of pressure of this order should then be experimentally assessed in animals and men.

Dissemination of External Pressure Throughout the Underlying Tissues

The distribution of pressure within gases, liquids, and rigid solids follows simple rules, but this is not so in the case of heterogeneous semi-solids such as human flesh. A simplified model made out of a foam rubber block can be used to illustrate some aspects of the problem. Such a model, with holes cut out to simulate blood vessels, is shown in figure 6. When the block is pressed upon from above by a solid object, then the area immediately beneath it is markedly compressed and the contained "vessels" are obliterated. This forceful compression, however, does not extend deeply, and it also must be noted that the areas adjacent to the impinging block are subjected not to compression but to increased tension. This tissue distortion is much greater when the impinging object is pointed

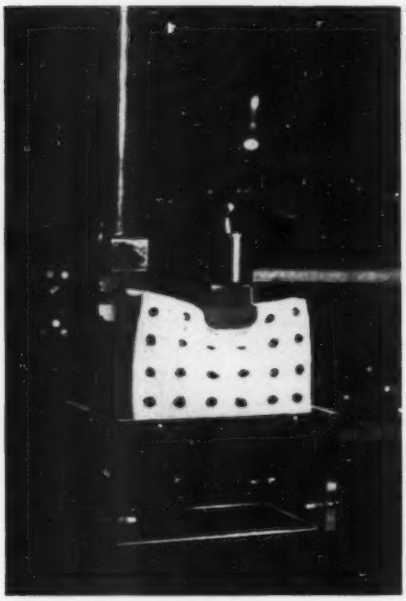


Fig. 6 — Foam rubber model to demonstrate the dissemination of localized external pressure within a compressible body.

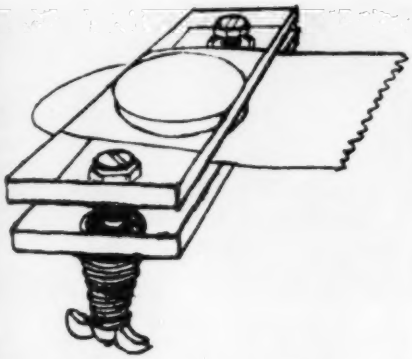


Fig. 7 — Enlarged model of the ear clip used to produce experimental pressure lesions on rabbits' ears.

Fig. 8 — Experimental rabbits with pressure clips in place on the ears.

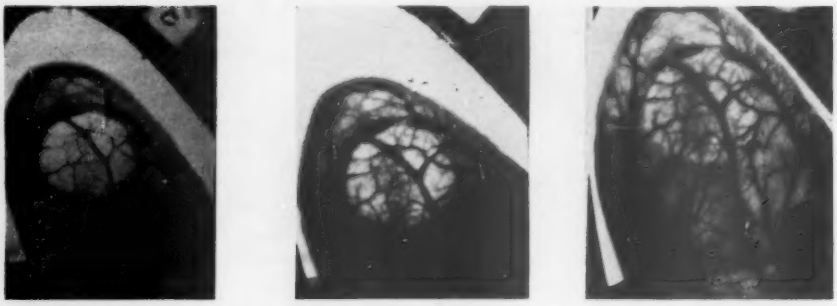
or angular, and when the pressure forces are not only vertical but shearing. Bony prominences will press on tissues in a similar way and cause varying degrees of distortion according to their shape and to the direction of pressure forces, which often are tangential to the skin.

In order to see how such a model compares with body tissues, a complete cross section of a limb was studied, using rows of pins as markers and observing the distortion through a plate of Plexiglass on which squares had been etched. Although flesh is not compressible like foam rubber, since there can be no escape of air, the muscle layers slide over one another and flow away from the point of immediate pressure, producing a picture of distortion fairly similar to that seen in the rubber models.

Vascular Response to Pressure in Animals

The question of what happens to living blood vessels subjected to varying degrees of pressure for various lengths of time is obviously of great importance in the study of decubiti. Rabbits' ears were chosen for the initial experiments in this field because gross changes in vascularity are easily visible and can be followed in the living animal.

Figure 7 shows an enlarged model of the ear clip used to produce the required degree of pressure. The ear — represented in the figure by a piece of paper — is sandwiched between two plastic discs, and the desired pressure is obtained by adjusting the nuts and compressing the two springs to a predetermined length. In this way a measured pressure can be exerted, regardless of the thickness of the ear, to an accuracy of within 10 per cent. The pressure surfaces of these discs measure 3.6 cm.^{2} and the clips are applied over the distal end of the pinna. The ears



Figs. 9, 10 and 11 — The effect of 100 mm. Hg pressure applied for seven hours. Appearance of rabbit's ear after two, five, and 90 seconds after removal of pressure clip. Dark spots around the pressure circles are the ink marks used to define the areas.

were depilated and both skin and clips were disinfected before the application of pressure. A battery of such experiments in progress is illustrated in figure 8.

The initial series of experiments was performed on healthy rabbits using a *constant pressure of 100 mm. Hg* for varying lengths of time. This pressure completely obliterates blood vessels in the area.

When the clip was removed after seven to nine hours of continuous pressure, the tissue beneath was at first noted to be white and bloodless, with hyperemia in the surrounding tissue, but the blood then returned rapidly within a few seconds (figs. 9-11).

Half an hour later marked extravasation of blood elements and tissue edema was noted (fig. 12a).

Eighteen hours later the edema was further increased, as measured by the thickness of the injured area; but there was less inflammation in the surrounding tissues (fig. 12b).

After seven days evidence of vascular damage was still quite marked with the major vessels still dilated and atonic.

Fig. 12a — Appearance of rabbit's ear half an hour after removal of pressure clip.

Fig. 12b — Appearance of rabbit's ear 18 hours after removal of pressure clip.

Figs. 13 and 14 — Appearance of the ear seven and 12 days after application of pressure.

The hair did not grow over the injured area (fig. 13).

As late as 12 days after the application of pressure the vessels still showed evidence of damage (fig. 14), illustrating the very slow process of healing following injuries of this type, although there had been no ulceration or macroscopic break of the skin surface.

If complete anoxia produced by a pressure of 100 mm. Hg was maintained for three hours only, the initial sequence of vascular response was similar, but the ear recovered very quickly and after a couple of days there was no significant lesion to be seen.

It should be noted that throughout

Fig. 15 — The effect of 100 mm. Hg applied for 13 hours. Appearance of rabbit's ear 12 hours after removal of pressure clip. Photo on the left made with translucent illumination; photo on the right under direct light.

this series of pressure lesions no thrombosis of major vessels was observed. These experimental lesions produced after three to nine hours of pressure resemble to a certain extent the early pressure lesions seen in bed and wheelchair-bound patients, when the area of damaged skin is sharply limited, is bright red, and blanches on finger pressure. If no further pressure is exerted on such areas, these lesions usually heal well without progressing to ulceration.

When continuous pressure was maintained for 13 to 15 hours the damaging effect was correspondingly greater, and within a few hours following release of the clip a frank hemorrhage was visible within the injured area with a widespread inflammation surrounding it (fig. 15). Later necrosis occurred at the point of pressure with the formation and shedding of a dry scab.

Fig. 16 shows a magnified picture of a moderately injured area, demonstrating the dilated blood vessels, edema, and extravasation of blood elements characteristic of these pressure lesions.

Fig. 16 — Magnified picture of moderate pressure injury on a rabbit's ear.

These experiments, using the same level of pressure, demonstrate clearly the relationship between the duration of pressure and the degree of vascular injury. In summary it may be said that in the healthy rabbit whose ears have been subjected to pressure producing complete localized anoxia, the subsequent injury can be classified as slight after three hours of pressure, as moderate after seven to nine hours, and as severe after 13 to 15 hours. It also follows from this that, in order to determine the lowest pressure capable of producing a severe lesion, a minimum time of 13 hours of pressure has to be used.

Pressures of 20, 40, 60 and 100 mm. Hg were applied for a constant period of 13 hours to various ears in the second group of rabbits and the resulting lesions compared.

The pressure of 20 mm. Hg caused no grossly demonstrable occlusion of the blood vessels and, after a transient hyperemia following the removal of the clip, the ears returned to their normal macroscopic appearance after 24 hours.

Similarly, 40 mm. Hg did not cause appreciable blanching of the pressure areas but the vessels became engorged following removal of the clip and the outlines of the smaller vessels became obscured by the exudate within one hour. There was some edema persisting for about two days and the lesion gradually resolved over an average of seven days.

The initial response, within the first two days, to compression at a level of 60 mm. Hg was very similar to that produced by 100 mm. Hg. Following this, however, there was a marked variation in the subsequent progress of the lesions, both the rate of healing and the formation of scabs being quite inconsistent.

Thrombosis of the major vessels was noted in a few instances following the application of pressure at 40 and 60 mm. Hg.

It seems therefore, from these preliminary studies in healthy rabbits, that a necrotic lesion, reasonably consistent in character, can be produced by the application of a pressure of 100 mm. Hg over a period of 13 to 15 hours using clips of the type described.

Variations in response due to those contributory causal factors (metabolic, infective and neurologic) which can be experimentally produced in rabbits, can thus be evaluated by (a) keeping the pressure constant at 100 mm. Hg and applying it for periods shorter than 13 hours or by (b) using lower pressures over a constant period of 13 hours. On the basis of the findings in rabbits, similar experiments are being extended to other laboratory animals using both the ears and surgically produced skin flaps.

It is expected that the experimental approach, as described above, which allows the independent study of single contributory factors, will further elucidate the role played by each in the formation of decubitus ulcers.

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Information relative to securing reprints of this study may be had by checking the Reader Service column on page iv of this issue.

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The following rules and regulations apply:

1. Any subject of interest or pertaining to the field of physical medicine and rehabilitation may be submitted.

2. Manuscripts must be in the office of the American Congress of Physical Medicine and Rehabilitation, 30 N. Michigan Ave., Chicago 2, not later than March 2, 1962.

3. *Contributions will be accepted from medical students only.*

4. The American Congress of Physical Medicine and Rehabilitation shall have the exclusive right to publish the winning essay in its official journal, the *Archives of Physical Medicine and Rehabilitation.*

5. Manuscripts must not exceed 3000 words (exclusive of headings, references, legends for cuts, tables, etc.), and the number of words should be stated on the title page. An original and one carbon copy of the manuscript must be submitted.

6. The essay must not have been published previously.

7. The winner shall receive a cash award of $100.

8. The winner shall be determined by the Awards and Prizes Committee of the American Congress of Physical Medicine and Rehabilitation.

9. All manuscripts will be returned as soon as possible after the name of the winner is announced. The winning manuscript becomes the exclusive property of the American Congress of Physical Medicine and Rehabilitation.

10. The American Congress of Physical Medicine and Rehabilitation reserves the right to make no award if, in the judgment of the Awards and Prizes Committee, no contribution is acceptable. Announcement of the winner will be made at the annual meeting.

dca

Announcing . . .

The Ninth Essay Award

sponsored by the

AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION

To stimulate interest in the field of physical medicine and rehabilitation, the American Congress of Physical Medicine and Rehabilitation will award annually, a prize for an essay on any subject relating to physical medicine and rehabilitation. The contest, while open to anyone, is primarily directed to interns, residents, graduate students in the pre-clinical sciences and graduate students in physical medicine and rehabilitation. The Awards and Prizes Committee suggests that members of the American Congress and American Academy of Physical Medicine and Rehabilitation bring this announcement to the attention of interested persons. The following rules and regulations apply to the contest:

1. Any subject of interest or pertaining to the field of physical medicine and rehabilitation may be submitted.

2. Manuscripts **must be** in the office of the American Congress of Physical Medicine and Rehabilitation, 30 N. Michigan Ave., Chicago 2, not later than March 2, 1962.

3. Contributions will be accepted from interns, residents, graduate students in the pre-clinical sciences, and graduate students in physical medicine and rehabilitation.

4. The essay must not have been published previously.

5. The American Congress of Physical Medicine and Rehabilitation shall have the exclusive right to publish the winning essay in its official journal, the ARCHIVES OF PHYSICAL MEDICINE AND REHABILITATION.

6. Manuscripts must not exceed 3000 words (exclusive of headings, references, legends for cuts, tables, etc.), and the number of words should be stated on the title page. An original and one carbon copy of the manuscript must be submitted.

7. The winner shall receive a cash award of $200.

8. The winner shall be determined by the Awards and Prizes Committee of the American Congress of Physical Medicine and Rehabilitation.

9. All manuscripts will be returned as soon as possible after the name of the winner is announced.

10. The American Congress of Physical Medicine and Rehabilitation reserves the right to make no award if, in the judgment of the Awards and Prizes Committee, no contribution is acceptable. Announcement of the winner will be made at the annual meeting.

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